



THE BRITISH  
JOURNAL OF SURGERY





# THE BRITISH JOURNAL OF SURGERY

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# CONTENTS OF VOLUME XIX.

## NUMBER 73.

	PAGE
SOME BYGONE OPERATIONS IN SURGERY :—	
Lithotrixy : the Case of the Emperor Napoleon III - <i>Sir D'Arcy Power</i>	1
THE PRESERVATION OF THE ILEOÆCAL SPHINCTER IN RESECTION OF THE RIGHT HALF OF THE COLON - - - - - <i>W. H. Ogilvie</i>	8
CHRONIC FOLLICULAR GASTRITIS : WITH A REPORT OF NINE CASES <i>R. R. Fitzgerald</i>	25
CHONDROSARCOMA OF THE FIBULA - <i>A. Victor Neale and E. D. Allen-Price</i>	47
THE TIME FACTOR DURING WHICH VASCULAR RESPONSES PERSIST FOLLOWING PERI- ARTERIAL SYMPATHECTOMY - - - - - <i>Lambert Rogers</i>	52
PAPILLIFEROUS CYSTOMA OF THE PETROUS BONE ASSOCIATED WITH HYPERNEPHROMA AND CYSTIC PANCREAS - - - <i>Geoffrey Jefferson and Raymond Whitehead</i>	55
A CLINICAL STUDY OF ACUTE APPENDICITIS IN OLD AGE - - <i>J. Lewin</i>	63
A CONTRIBUTION TO THE SURGERY OF THE PITUITARY REGION. AN ACCOUNT OF FOUR CASES OF 'PITUITARY TUMOUR' TREATED BY RADON SEEDS <i>L. Bathe Rawling</i>	68
TRAUMATIC ARREST OF EPIPHYSIAL GROWTH AT THE LOWER END OF THE TIBIA <i>Bryan McFarland</i>	78
CARCINOMA OF THE THYROID GLAND - - - - - <i>T. P. Dunhill</i>	83
THE PATHOLOGY AND TREATMENT OF TUMOURS OF THE CAROTID BODY <i>H. Hamilton Stewart</i>	114
THE OPERATIVE TREATMENT OF POTT'S PARAPLEGIA - <i>G. R. Girdlestone</i>	121
SIALOGRAPHY : ITS TECHNIQUE AND APPLICATIONS - - <i>Reginald T. Payne</i>	142
TWO CASES OF CARCINOMA OF THE SMALL INTESTINE <i>O. Herbert Williams, W. Robert Williams, and R. Howard Mole</i>	149
SHORT NOTES OF RARE OR OBSCURE CASES - - - <i>Various Writers</i>	153
REVIEWS AND NOTICES OF BOOKS - - - - -	159

## NUMBER 74.

SOME BYGONE OPERATIONS IN SURGERY :—	
Amputation : the Operation on Nelson in 1797 - <i>Sir D'Arcy Power</i>	171
STRANGULATED HERNIA : A REVIEW OF 1487 CASES - <i>Claude Frankau</i>	176

	PAGE
A CASE OF GENERALIZED FIRROCYSTIC DISEASE OF THE BONES <i>Lieut.-Colonel E. W. C. Bradfield</i>	192
HYPERPARATHYROIDISM: GENERALIZED OSTEITIS FIBROSA. (WITH OBSERVATIONS UPON THE BONES, THE PARATHYROID TUMOURS, AND NORMAL PARATHYROID GLANDS BY <i>Hubert M. Turnbull</i> ) - - - - <i>Donald Hunter</i>	203
THE SURGICAL TREATMENT OF PARATHYROID TUMOURS - <i>A. James Walton</i>	285
'MADE-TO-ORDER' SPLINTS: THE TECHNIQUE OF CONSTRUCTION AND FIELD OF APPLICATION - - - - - <i>Hugh Trumble</i>	292
REGENERATION OF THE INTERNAL SEMILUNAR CARTILAGE AFTER OPERATION <i>A. Gibson</i>	302
EXPOSURE OF THE KNEE-JOINT - - - - - <i>H. B. Devine</i>	306
CHOLECYSTITIS GLANDULARIS PROLIFERANS (CYSTICA) <i>E. S. J. King and P. MacCallum</i>	310
VISITS TO SURGICAL CLINICS AT HOME AND ABROAD:— Dr. Mink Jansen and his Clinic at Leiden - - <i>Alan H. Todd</i>	324
SHORT NOTES OF RARE OR OBSCURE CASES - - - <i>Various Writers</i>	330
REVIEWS AND NOTES OF BOOKS - - - - -	341

## NUMBER 75

SOME BYGONE OPERATIONS IN SURGERY:— Amputation: the Operation on Nelson in 1797 - <i>Sir D'Arcy Power</i>	351
CONGENITAL LYMPHANGIECTATIC FIBROUS HYPERTROPHY (ELEPHANTIASIS CON- GENITA FIBROSA LYMPHANGIECTATICA) - - - <i>D. Stewart Middleton</i>	356
TWO CASES OF LOCALIZED PHLEGMONOUS ENTERITIS <i>C. Gordon Irwin and Stuart McDonald, Junr.</i>	362
SPONDYLOLISTHESIS - - - - - <i>Norman Capener</i>	374
POST-OPERATIVE MASSIVE COLLAPSE OF THE LUNG. A CLINICAL AND EXPERIMENTAL STUDY - - - - - <i>David Baud and I. Simson Hall</i>	387
AN ANALYSIS OF 2126 CASES OF ACUTE APPENDICITIS <i>B. R. Sworn and G. M. Fitzgibbon</i>	410
THE RADIUM TREATMENT OF CARCINOMA OF THE BREAST - <i>Geoffrey Keynes</i>	415
ON THE EFFECT OF ANÆMIA ON THE REACTIONS OF THE SKIN AND OF TUMOURS TO RADIUM EXPOSURE - - - - <i>J. C. Mottram and Albert Eidinow</i>	481
THE PATHOLOGY AND TREATMENT OF TUBERCULOSIS OF THE KNEE-JOINT <i>G. R. Girdlestone</i>	488
THREE CASES OF TORSION OF AN APPENDIX EPILOICA OF THE SIGMOID COLON <i>G. H. Colt</i>	508
SHORT NOTES OF RARE OR OBSCURE CASES - - - <i>Various Writers</i>	511
REVIEWS AND NOTICES OF BOOKS - - - - -	515

## NUMBER 76.

SOME BYGONE OPERATIONS IN SURGERY:—		
The First Localized Cerebral Tumour	- - -	<i>Sir D'Arcy Power</i> 523
DIAPHRAGMATIC HERNIA	- - -	<i>J. Basil Hume</i> 527
THE TREATMENT OF DUPUYTREN'S CONTRACTURE	- - -	<i>A. A. Davis</i> 539
URETEROCELE: SOME OBSERVATIONS BASED ON THE INVESTIGATION AND TREATMENT OF FOUR CASES	- - -	<i>J. C. Ainsworth-Davis</i> 548
THE ADRENO-GENITAL SYNDROME ASSOCIATED WITH CORTICAL HYPERPLASIA; THE RESULTS OF UNILATERAL ADRENALECTOMY		
	<i>L. R. Broster, H. Gardiner Hill, and J. G. Greenfield</i>	557
EMBRYONIC EPITHELIAL RESTS IN THE PITUITARY	- - -	<i>William Susman</i> 571
ISOLATED FRACTURE OF THE CARPAL SEMILUNAR AND KIENBÖCK'S DISEASE		
	<i>T. B. Mouat, John Wilkie, and H. E. Harding</i>	577
HYDATID PNEUMOTHORAX	- - -	<i>Sir Louis Barnett</i> 593
ON SOME COMPLICATIONS OF TRAUMATIC DISLOCATION OF THE HIP-JOINT		<i>Harry Platt</i> 601
A CASE OF PARATHYROID TUMOUR ASSOCIATED WITH FIBROCYSTIC DISEASE		
	<i>G. Gordon-Taylor, Philip Wiles, and S. L. Baker</i>	606
THE INCIDENCE OF ANAEROBIC INFECTIONS IN THE GALL-BLADDER		
	<i>G. Gordon-Taylor and Lionel E. H. Whitby</i>	619
FURTHER STUDIES IN INTRAVENOUS PYELOGRAPHY	<i>R. J. Willan and James H. Saint</i>	622
THE SYMPATHETIC IN ACUTE GENERAL PERITONITIS. A CLINICAL STUDY WITH OBSERVATIONS ON TREATMENT		
	<i>John J. Robb</i>	634
A CASE OF RAPIDLY-GROWING CARCINOMA IN THE NECK, ARISING IN A PARATHYROID REST		
	<i>L. Woodhouse Price and G. T. Morat</i>	645
THE OPERATIVE TREATMENT OF THE ELEPHANTOID SCROTUM	<i>W. K. Connell</i>	651
SHORT NOTES OF RARE OR OBSCURE CASES	- - -	<i>Various Writers</i> 659
REVIEWS AND NOTICES OF BOOKS	- - -	661

## GENERAL INDEX TO VOLUME XIX.

### SUPPLEMENT: ATLAS OF PATHOLOGICAL ANATOMY.

FASCICULUS VII:	{	DISEASES OF THE THYROID GLAND.
		DISEASES OF THE MOUTH, PHARYNX, AND ŒSOPHAGUS.



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## *SOME BYGONE OPERATIONS IN SURGERY.*

By SIR D'ARCY POWER, K.B.E., LONDON.

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### V. LITHOTRITY:

#### THE CASE OF THE EMPEROR NAPOLEON III.

LITHOTRITY in its original form had a short and brilliant career in the history of surgical operations. In a modified form it is still in use, but with diminishing frequency, at any rate in England, where stone in the bladder is becoming yearly less common, as hygiene, dietetics, and social habits improve, with advances in our knowledge of the laws of health.

Lithotrity began in the early part of the last century as a counterblast to the use of lithontryptics and lithotomy, which were yielding very unsatisfactory results. Jean Civiale (1792-1867), when he was as yet only a second-year medical student in the University of Paris, began a series of experiments in 1817 to ascertain whether it was possible to crush a stone in the bladder without injuring the walls. He invented a variety of instruments for the purpose and read many papers before the scientific societies, but it was not until 1823 that he ventured to operate upon a living patient. His method did not attract much attention at first outside Paris, though Jean Leroy (D'Étiolles), the tactless and jealous Charles Louis Stanislas Heurteloup, and Franz von Gruithuisen, who was afterwards Professor of Astronomy at Munich, were also devising instruments for the same purpose.

The earlier instruments were large and formidable pieces of apparatus, consisting in principle of a straight metal catheter provided with a claw-shaped end by which the stone was seized and held in position whilst it was being broken up by a metal rod passed through the catheter. The stone was either pierced, drilled, crushed, or squeezed by force exerted through the rod. An attempt was made to crush the stone with these instruments at a single sitting, the fragments being allowed to pass by the natural expulsive efforts of the bladder.

The method spread slowly beyond France, and it was not until 1827 that lithotrity was undertaken in Austria and in Italy. Baron Heurteloup came to London in 1828 and lived in Vere Street until 1832. On July 24 and 30 and on August 20, 1829, he operated upon a seaman, age 64, who was under



the care of Anthony White in Westminster Hospital, and this was the first case of lithotrity in England. Heurteloup had improved his technique by 1832, for he was then crushing stones at a single sitting and removing the fragments by syringing out the bladder through a catheter with a large eye. He claimed that for this purpose he invented the syringe now in general use which has three rings to enable it to be used with one hand.



SIR HENRY THOMPSON

The value of the operation was quickly recognized. Joseph Hodgson in England and Philip Crampton in Dublin performed it frequently, simplified the instrument, and improved the method of removing the fragments, though they preferred to crush the stone at repeated sittings lasting only a few minutes. The lithotrite by this time was assuming its present shape—a rod ending in a flattened blade set at an angle. A movable blade passed along the fixed one,

the sliding movement being converted into a screw motion when the stone had been seized. The female blade, at first solid, was afterwards fenestrated to allow the fragments of the stone to drop back into the bladder. The standardization of the instrument was due largely to the mechanical genius of John Weiss, the instrument maker, who then had a shop in the Strand. Credit must also be given to Charrière, of Paris, whose model Weiss improved in several particulars.



PROFESSOR H. JACOB BIGELOW

The practice of lithotrity soon fell largely into the hands of (Sir) Henry Thompson, who was devoting himself to the surgical treatment of diseases of the urinary organs. Thompson visited Paris in July, 1858, to study under Civiale, and in 1863 he published the results of his experience in the Lettsomian Lectures delivered before the Medical Society of London. Improvements had in the meantime been made in the method of evacuating the fragments of

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crushed stone, and in 1846 Philip Crampton was using as a suction apparatus a glass bottle exhausted of air and connected with a catheter by means of a rubber tube provided with a stopcock. Mr. J. T. Clover, the anæsthetist, invented his apparatus in 1866, which, with slight modifications, is still in use. It consists of a rubber bulb with a glass trap to catch the fragments as they are washed out through an evacuating catheter.

The stone was crushed at repeated sittings until in 1878, when Professor H. Jacob Bigelow (1818–1890) of the Harvard University, Boston, Mass., revolutionized the operation by crushing the stone at a single sitting (continued for as long as might be necessary) and removing all the fragments whilst the patient was still under the anæsthetic. He did this by using a more powerful lithotrite, a larger evacuating catheter, and a more efficient evacuating apparatus. The operation was carefully thought out and was the result of more than three years' experimental work. His first operation was performed on May 15, 1876, the patient being a man of 60 who had suffered with symptoms of stone for twenty years. The method of litholapaxy or rapid lithotrixy with evacuation was brought to the notice of English surgeons by Reginald Harrison at the annual meeting of the British Medical Association on August 8, 1878, but it did not come into use for several years afterwards.

The success of Sir Henry Thompson's lithotrities, many of which were done at University College Hospital, became so well known that in 1868 he was called upon to complete the work which Civiale had begun eighteen months previously and crush the stone which had long troubled Leopold I, King of the Belgians. The earlier sittings had been suspended owing to an attack of acute cystitis, but Thompson avoided its recurrence by using a new lithotrite on each occasion. Nine years later he was summoned to Camden Place, Chislehurst, in Kent, to consider the case of the Emperor Napoleon III. The Emperor had suffered from urinary trouble for some years, and on July 1, 1870, a consultation had been held at the Tuileries to determine the cause of his sufferings. There were present MM. Nélaton, Ricord, Fauvel, Sée, Corvisart, and Conneau, the last being his private medical attendant. Professor Sée was asked to put the findings of the consultants into writing, and Conneau undertook to obtain the signatures of the surgeons and bring the report to the notice of the Empress. The report, dated July 3, 1870, stated that the Emperor had suffered from four attacks of hæmaturia in the preceding five years and that from August, 1869, the urine had always contained pus. The shaking of a carriage and riding on horseback caused much pain. There was also dysuria, and a catheter had been used on two occasions at least. It was thought from these signs and symptoms that the Emperor undoubtedly had a renal or a vesical calculus, and exploratory measures were recommended. The report, however, miscarried owing no doubt to the turmoil prevailing in those July days just before the declaration of war against Germany. It was never signed by the consultants and it was never shown to the Empress. It seems to have been kept by Dr. Conneau, for it was seized with his other papers at the Tuileries on September 4, 1870, and was then published. The document is of historical importance owing to the date on which it was drawn up—July 3, 1870—three days before the declaration of war.

Had it been seen by the Empress and had an operation been decided upon, events might have happened very differently. The outbreak of hostilities might have been delayed or even entirely abandoned. As it was, they were undertaken by a very sick man, and in the end Napoleon came to England as an exile.

The symptoms improved for some time after the arrival of the Emperor in England from Wilhelmshöhe, but later they increased in severity until in July, 1872, Sir William Gull and Sir Henry Thompson met Dr. Conneau in consultation. The prostate was then found to be healthy, but the Emperor declined to allow a catheter to be passed. Palliative measures, therefore, could alone be recommended. The pain increased, and prevented riding, driving, and even walking, until on October 31 he consulted Sir James Paget, who advised an exploration of the bladder. This was again declined, and it was not until December 24 that Sir Henry Thompson was allowed to pass a flexible catheter, after which he impressed upon the patient the urgent necessity for an exploration of the bladder under an anæsthetic.

The necessary arrangements were made, and on Thursday, January 2, 1873, an anæsthetic was given by Mr. Clover. Sir Henry Thompson introduced a lithotrite and determined the presence of a vesical calculus, probably phosphatic, of the size of a walnut or large chestnut. The examination was made at 3.30 p.m. in the presence of Sir William Gull, Baron Corvisart, Dr. Conneau, and Mr. John Foster (brother of Sir Michael Foster the physiologist), who was then acting as Thompson's private assistant. The stone was crushed and a considerable quantity of débris was removed. The operation was followed by much pain, and a second sitting was arranged for Monday, January 6, at 10 a.m. This second sitting had to be postponed for two hours as the patient had a rigor, and it was not until 12 o'clock midday that the anæsthetic was administered, again by Mr. Clover. A large fragment of stone was then found impacted in the membranous portion of the urethra. Much delicate handling had to be undertaken before it was dislodged and further crushing was possible. There was evidence of obstruction on the day following this second sitting, but it was less complete, and it was decided not to pass any instrument. A third séance was arranged for the morning of Thursday, January 9, but by this time it is evident that the general condition of the Emperor had given rise to anxiety. He was seen at 11 o'clock on Wednesday night by his medical attendants; at 2 a.m. on Thursday by Dr. Conneau; at 4 a.m. by Baron Corvisart; at 6 a.m. by Sir Henry Thompson; and at 9.45 a.m. by all his medical attendants in consultation, as well as by Mr. Clover.

It was reported that the Emperor had slept better than on the previous night, and it was decided to proceed with a further lithotrity at 12 o'clock. He became alarmingly ill, however, at 10.25, and died at 10.45. A post-mortem examination was made by Dr. J. Burdon Sanderson, then the foremost pathologist of the day, who found extensive disease of both kidneys. The calculus weighed about  $\frac{3}{4}$  oz. and measured  $1\frac{1}{4} \times 1\frac{1}{8}$  in. All the other organs were healthy. About one-half of it had been crushed. Its nature is not stated, but it appears to have consisted of urates with a coating of phosphates.

Sir Henry Thompson, the operator, was a remarkable man. Born in 1820, he was the only son of the village shopkeeper at Framlingham in Suffolk, by his wife Susannah, daughter of Samuel Medley, the artist who painted the well-known portrait group of the founders of the Medical Society of London. Medley afterwards went on the Stock Exchange, made money, and was instrumental in founding University College (afterwards the London University) in 1826. Both the parents of Sir Henry Thompson were rigid noneonformists who dreaded a scientific education and disliked the idea of a profession for their son. Thompson, therefore, was employed for a short time in his father's business, but soon tiring of it, apprenticed himself to George Bottomley, a medical man practising at Croydon in Surrey. He entered as a medical student at University College, London, in October, 1844, and distinguished himself by winning the gold medals in anatomy and surgery and by his appointment under Sir John Eric Erichsen as his first house surgeon. At the end of his term of office he entered into partnership with his former master George Bottomley, but soon returned to London with the intention of practising as a surgeon. He was elected Assistant Surgeon to University College Hospital in 1853, becoming full Surgeon in 1863, Professor of Clinical Surgery in 1866, and Consulting Surgeon in 1874.

Thompson was an ardent champion of cremation as a method of disposal of the dead. He first drew attention to the subject by an article in the *Contemporary Review* in 1874, and by his energy established a Cremation Society of which he remained President until his death. A Crematorium was built at Woking, but its use was forbidden by Lord Cross, who was then Home Secretary, and it was not until March, 1885, that the first cremation was undertaken after Sir James Stephen had declared in a test case that the procedure was not illegal if it were carried out decently and without offence.

Thompson was a skilled artist, the talent no doubt being derived through his mother, though it was fostered by study under Edward Elmore, R.A. and Sir Lawrence Alma Tadema, and he exhibited frequently at the Royal Academy. He was also interested in astronomy and built an observatory at Molesey, where he had a country house. He was, too, an eminent collector of china, his collection being sold at Christie's on June 1, 1880. In the social life of London he played a great part as a host. He was celebrated for his 'Octaves'—dinners of eight persons, eight courses, at 8 o'clock. The company, the food, and the wines were of the choicest. King Edward was of the party on more than one occasion, and King George V, when Prince of Wales, was present at the 300th octave. Thompson received the honour of knighthood in 1867 and was created a baronet in 1898. He married Kate Fanny, daughter of George Loder, of Bath, and was the father of a son, Sir Henry Francis Herbert Thompson, and two daughters. Lady Thompson was well known for her musical talent as a pianist.

Sir Henry Thompson died at 35 Wimpole Street, where he had lived all his professional life, on April 18, 1904, and his body was cremated at the Golder's Green crematorium.

He wrote much, both professionally and in the lay reviews and magazines, and published two novels under the name of "Pen Oliver, F.R.C.S.," the

first in 1885 being entitled *Charlie Kingston's Aunt*, the second in 1886, *All But, a Chronicle of Laxenford*, which is illustrated by twenty-four whole-page drawings by himself.

The portrait of Sir Henry Thompson is reproduced from *All But*, a copy of which was kindly lent by Mr. George Buckston Browne, F.R.C.S.Eng., who was formerly his private assistant. The original was drawn by Sir Henry Thompson, and represents him as he appeared in 1885 in Framlingham, his native village. The portrait of Professor Bigelow is from a photograph taken in 1888.



## THE PRESERVATION OF THE ILEOCÆCAL SPHINCTER IN RESECTION OF THE RIGHT HALF OF THE COLON.

By W. H. OGILVIE,

ASSISTANT SURGEON, GUY'S HOSPITAL, LONDON.

### REASONS FOR EXCISION OF THE ILEOCÆCAL SPHINCTER.

RESECTIONS of the colon are performed, in the great majority of cases, for the removal of malignant growths. In such an operation the growth itself, as much of the bowel above and below and of neighbouring structures as may conceivably have been involved by cancerous cells, and the whole of the lymphatic vessels and glands which drain the affected area, must be removed in one block. Since the lymphatic channels from the colon accompany the blood-vessels, and the intermediate and main lymphatic glands are grouped along them, any such radical operation implies the ligature of the corresponding colic artery shortly below its origin from the superior or inferior mesenteric trunk. Thus a second factor arises: the parts removed must include not only those necessary for eradication of all the areas which may have undergone malignant invasion, but also those whose viability has been endangered by ligature of the main artery. In the case of growths lying anywhere between the ileocecal valve and the hepatic flexure, these considerations demand the removal of the area supplied by the ileocolic and right colic arteries, and therefore a resection which is sometimes wider than is necessary upon grounds of pathology alone.

Friedrich's operation, resection of the intestine from a point six inches above the ileocecal valve to a point four inches below the hepatic flexure, with the peritoneum overlying the ileocolic and right colic arteries and the lymphatics accompanying them, has for many years been accepted as an eminently satisfactory procedure for the treatment of operable cancer in the right half of the colon. The immediate and late results of Friedrich's operation as regards safety and prospects of cure are admirable, but it implies the removal of one of the most important pieces of mechanism in the physiology of digestion, the ileocecal sphincter. In cancer surgery this is unavoidable, for the need for eradication of the disease overrules all other considerations, and cancer surgery has hitherto been dominant. Of late, however, operations upon the colon for the treatment of non-malignant conditions have come to assume an increasing importance. Diverticulitis, stasis in the proximal colon, and dysfunction after suppurative appendicitis, may all demand resections of the right half of the colon in which any wide removal of the lymphatic drainage area is unnecessary. The purpose of this paper is to suggest that in such operations the physiology of the ileocecal angle should receive greater consideration.

## THE STRUCTURE, MOVEMENTS, AND FUNCTION OF THE ILEOCÆCAL SPHINCTER.

The observations of Keith in 1903 and of Rutherford in 1914 have established the fact that the ileocæcal opening is a muscular sphincter, which does not depend in its action upon any valvular mechanism. In life the termination of the ileum is seen as a hemispherical papilla about 1·8 cm. in diameter and projecting about 1 cm. above the surrounding cæcal wall: it is smooth and scarlet, in contrast to the pink and folded cæcal mucosa. The frænula described in anatomical text-books are never seen. The sphincter is formed by the fusion of the circular and longitudinal fibres of the termination of the ileum and those of the adjacent cæcum; in addition some independent circular fibres are grouped round its summit. The ileocæcal sphincter derives its nerve-supply from the sympathetic system. Section of the splanchnic nerves is followed by permanent relaxation; on the other hand, direct stimulation of these nerves, or indirect stimulation by adrenalin, produces violent contraction of the summit of the sphincter, while the movements of the rest of the intestine are inhibited. Stimulation of the vagi or pelvic nerves has no effect. Keith has demonstrated that the nodal tissue forming Auerbach's plexus is especially developed at the level of the ileocæcal sphincter, as at the cardia.

Rutherford has described the movements of the ileocæcal sphincter, as observed through a cæcostomy opening. During periods of activity, waves of peristalsis can be seen through the relaxed cæcal wall progressing along the terminal ileum at regular intervals. When the wave of contraction reaches the cæcum, the sphincter relaxes, and the papilla becomes wider and less prominent. About 4 c.c. of liquid fæces and gas enter the cæcum, and the sphincter then shuts and becomes firmer and less prominent.

The chief function of the ileocæcal sphincter is to prevent the ileal contents passing too rapidly into the cæcum. Hurst has shown that a barium meal reaches the end of the ileum one hour before any appreciable quantity enters the cæcum. The ileum is often full four or five hours after the last traces of a meal have left the stomach. In consequence the products of digestion collect in the terminal ileum, where they remain for a longer time than in the stomach. During this period active segmentation can be observed in the terminal four inches of the ileum, but peristalsis is always very feeble. The ileocæcal sphincter has therefore the same effect on the contents of the terminal ileum as the pyloric sphincter has on the contents of the stomach, controlling the outflow, so that sufficient time may elapse for the complete digestion and absorption of foodstuffs. This physiological ileal stasis is so well regulated that the chyme which enters the cæcum only contains very small quantities of nutritive material in solution.

The ileocæcal sphincter relaxes whenever a wave of peristalsis passes over the terminal coils of the ileum. Though these waves start shortly after the arrival of the chyme at the ileocæcal angle, they are at first very infrequent. As a result of the ileocæcal reflex described by Hurst, active peristalsis takes place at the lower end of the ileum each time that a meal enters the stomach; the sphincter relaxes, and chyme enters the cæcum. The sphincter is not

called upon under normal conditions to prevent reflux of faecal matter into the ileum, since antiperistalsis does not occur in the proximal colon unless there is some distal obstruction. When such movements occur, the sphincter cannot withstand any degree of increased pressure on its colic surface.

**Spasm and Incompetence of the Ileocaecal Sphincter.**—Hurst believes that ileal stasis is rarely encountered except under conditions of organic obstruction at the ileocaecal angle. When true ileal stasis is seen in the absence of organic obstruction it is usually due to achalasia of the ileocaecal sphincter determined by a chronically infected appendix. This condition is exactly comparable to that of pyloric achalasia in duodenal ulcer.

Incompetence of the sphincter does not occur as a clinically observed phenomenon. After ileocolostomy with or without resection of the terminal ileum, the controlling action of the ileocaecal sphincter is entirely abolished and the contents of the ileum pour straight into the colon. The lower four feet of the small intestine become dilated, their muscular coats hypertrophy, and their lumen contains semi-solid faecal matter. The terminal stages of intestinal digestion and absorption are cut short, and much of the nutritive material in the chyme is lost to the body. Much more serious is the constant presence of the putrefactive bacteria in the small intestine, where they find abundant material on which to multiply, and where the free blood-supply and specialized absorptive function of the mucous membrane facilitates the entry into the circulation of the toxins which they produce.

The resection or exclusion of the ileocaecal sphincter for intestinal stasis in young patients is fraught, in the majority of cases, with disastrous consequences. At first, if constipation was very marked before the operation, the patients may feel much better, and this improvement may last for as long as five years. Later, progressive ill health, suggesting absorption of toxins, sets in. Irritability, nervousness, insomnia, a feeling of extreme weakness and tiredness, headaches, nausea, loss of appetite, and loss of weight are the main symptoms. No treatment has much effect in alleviating these troubles.

### THE OPERATION OF TRANSPLANTATION OF THE ILEOCAECAL SPHINCTER.

Any modification in technique which enables the essential object of an older operation to be attained, and at the same time preserves to a greater extent the function of the part, is a step forward. The operation of transplantation of the ileocaecal sphincter here described is an example of such an attempt to retain function. The immediate results of the operation in the two cases in which it has been carried out have been so satisfactory that they justify the publication of a method which has only had such brief trial. The principle of the operation was suggested by Dr. A. F. Hurst, who had both patients under his care in New Lodge Clinic. Full clinical details of the condition for which the operation was performed are appended at the end of this paper. The actual procedure was the same in each case.

The abdomen is opened by a right paramedian incision, 6 or 7 in. long,

## TRANSPLANTATION OF ILEOCÆCAL SPHINCTER 11

and about an inch from the mid-line, the rectus being displaced outwards. After separation of adhesions, and a general survey of the abdominal viscera, the peritoneum is divided to the outer side of the ascending colon (*Fig. 1*). This division is made at the edge of the bowel, or slightly overlapping it, so that plenty of peritoneum shall be available at the end of the operation for covering the whole posterior abdominal wall, and is carried upwards round the hepatic flexure for 2 in. along its superior border, and downwards round the attachment of the cæcum in the right iliac fossa, to continue for 1 in. along the inferior aspect of the attachment of the mesentery of the ileum. The cæcum, ascending colon, and hepatic flexure are mobilized by stripping them inwards as in the first stages of Friedrich's operation. In this conservative operation, however, the mobilization does not extend inwards to the



FIG. 1.—Division of peritoneum and mobilization of colon.

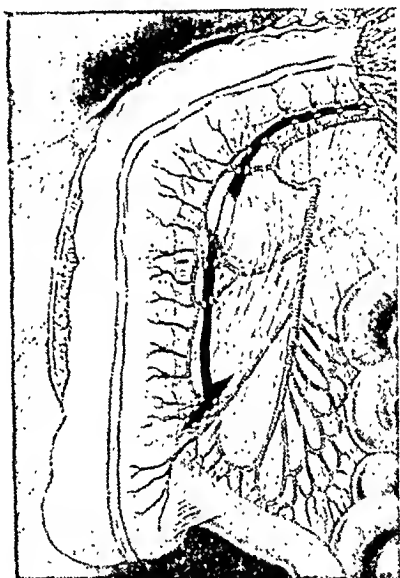


FIG. 2.—Division of vessels and isolation of ileocolic bundle.

ureter, and the stripping is less vigorous, the retroperitoneal layer of fascia being left on the muscles.

The mobilized colon is now held up, and the ileocolic artery and its branches are identified through the translucent peritoneum: the branches going to the right are divided between double ligatures about half an inch from the main ileocolic artery (*Fig. 2*), and the peritoneum is incised along the line of this division from the inner margin of the cæcum for about  $1\frac{1}{2}$  in. mesially. The blood-supply to the ileocolic angle, and the accompanying sympathetic plexus which innervates the sphincter, are thus left intact. Division of the peritoneum is carried upwards on the inner side of the ascending colon at a distance of about an inch from its wall, the right colic artery or its branches being cut between double ligatures. A point from one-third to one-half the distance along the transverse colon from the hepatic flexure is now selected for division, such a length being left as will allow the blind proximal end of

the divided colon to lie comfortably at the old site of the hepatic flexure without tension, yet without looping of the remaining transverse colon. The branches of the middle colic are divided close to the wall of the colon for a

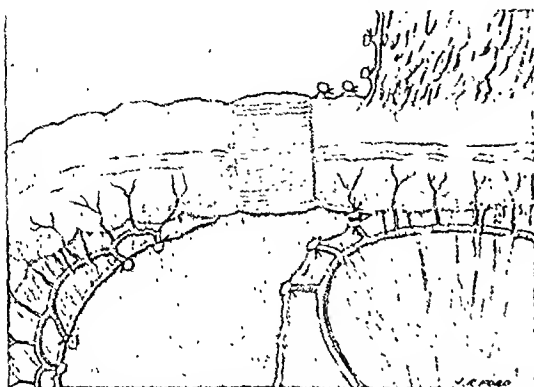


FIG. 3.—Transverse colon crushed at point selected for division.

distance of 1 in. on each side of the selected spot, and the attachments of the great omentum to the superior border of the part of the colon to be removed are tied off close to the bowel and divided. The colon is crushed with a powerful clamp 1 in. wide (*Fig. 3*), and tied in the crushed portion by two ligatures,  $\frac{1}{4}$  in. apart, which are left long. Proximal and distal to these ligatures, two purse-string sutures are placed round the bowel but not tied (*Fig. 4*). The colon is now divided between the ligatures with a cautery, the ends are held up by an assistant, and invaginated as the purse-string sutures are tightened by forceps which are immediately put aside. The blind end of the distal colon is further closed by a second purse-string suture.

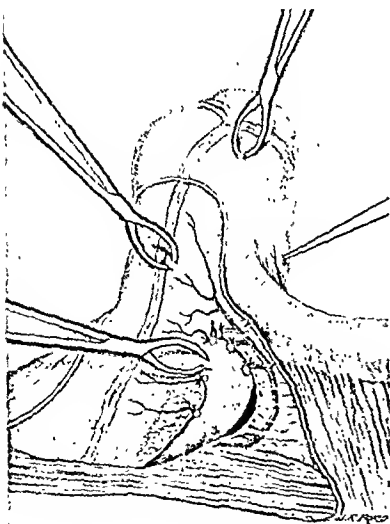


FIG. 5.—Diamond-shaped area on the medial caecal wall marked out for transplantation. (In the figure the ascending colon is rotated clockwise through  $120^\circ$  to bring the ileocaecal region towards the observer.)

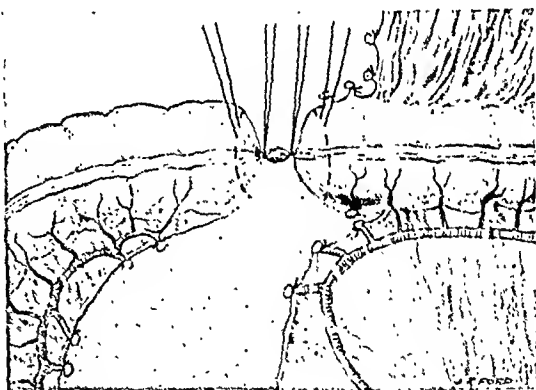


FIG. 4.—Transverse colon ligatured in crushed portion: two purse-string sutures in position, ready for tying.

The caecum is now prepared for anastomosis. The posterior wall has no peritoneal coat, but is covered with loose fat, in which the caecal vessels run before they pierce the muscular layer. All fat and connective tissue is cleared away along the line of suture, and any small vessels which are divided in the process are ligatured. A diamond-shaped piece of the medial caecal wall (*Fig. 5*), with the ileocaecal junction

at its centre, is then marked out for transplantation. The longer diagonal of the diamond, which lies in the long axis of the bowel, is 3 in. long, the shorter, at right angles to this, is 2 in. This area is marked out by three Lane's forceps attached at its upper, lower, and posterior angles to the cæcal wall, and a stitch at its anterior angle. The transverse colon immediately beyond the closed end is now emptied of contents by gentle pressure, and a loop 5 in. long and including about half its circumference is held in an intestinal clamp so that the anterior longitudinal band lies along its summit.

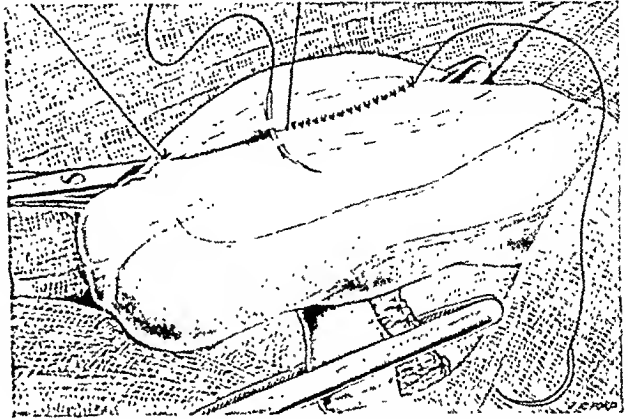


FIG. 6.—Cæcum brought up to transverse colon : insertion of first row of stitches.

The ileum is controlled by a light clamp placed across it 3 in. from its termination. The cæcum is brought up to the loop of transverse colon, with its posterior surface towards the colon, and its blind end

to the right (*Fig. 6*). Before the anastomosis is commenced, a strip of gauze soaked in flavine solution is laid between the two portions of bowel behind the line of junction, and abdominal mops, also wrung out in flavine, are arranged round the operation area to prevent any possible soiling.

The posterior cæcal wall is laid along the loop of colon, and the points marked by Lane's forceps are attached by three stitches of 00 catgut, the

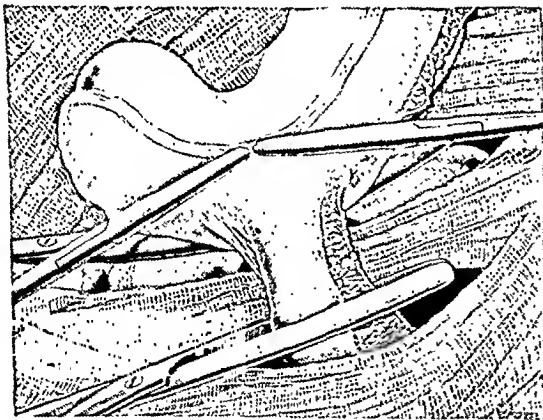


FIG. 7.—Posterior half of first row of stitches completed. Cæcum clamped off prior to removal.

upper and lower angles of the diamond being stitched to the longitudinal band, the posterior angle  $\frac{1}{2}$  in. below it. The stitch at the left-hand end of the anastomosis is left long, with the needle still on it. The cæcal wall between these stay sutures is then fixed to the transverse colon by a series of interrupted stitches about  $\frac{1}{8}$  in. apart. The cæcum is now emptied by pressure, and two long Ochsner forceps are placed across it from the inner side, one above and one below the ilioecæcal angle (*Fig. 7*): these

forceps outline a diamond slightly larger than that marked out by the stitch and Lane's clamps, their points meeting in the middle of the cæcum. A knife is run along the ilioecæcal side of these forceps, and the colon, closed

at one end by a purse-string suture, and at the other by the two clamps, is removed.

The exposed surface of the cæcum and ileocaecal valve is rapidly mopped dry and swabbed with gauze soaked in flavine solution. The transverse colon is opened by an incision along the longitudinal band, and its interior also

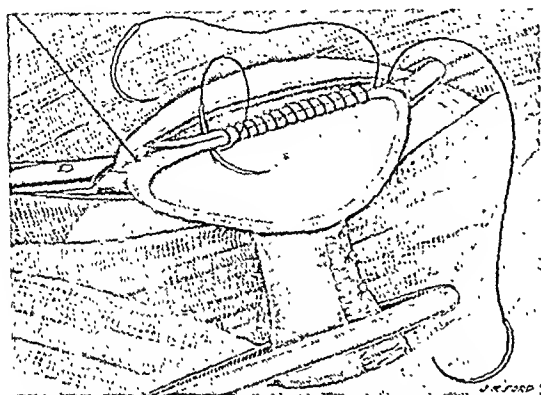


FIG. 8.—Cæcum removed, exposing ileocaecal sphincter. Insertion of first half of hæmostatic suture.

mopped clean and swabbed with flavine (*Fig. 8*). A stitch of 00 catgut is next inserted at the right-hand corner of the anastomosis, the needle piercing the transverse colon from the mucosa outwards and re-entering the cæcum from without inwards; this stitch is tied on the mucosa and left long. A second stitch, piercing all the coats of the adjacent edges of transverse colon and cæcum, is inserted half-way along the line of anastomosis, tied, and left long. The continuous hæmostatic suture is then started at

the left-hand end of the opening. This suture is of 00 catgut with a needle at each end, and is inserted in the same way as that at the right-hand corner, the knot being tied on the mucosa at the centre of the catgut. It is continued as an over-and-over stitch through all the layers, the first and second stay sutures, when reached, being knotted to the continuous one, and cut short. The return layer of the hæmostatic suture is done by a simple over-and-over stitch with pull on the mucosa which I learned five years ago from Tyrrell-Gray, and have since used for all anastomoses (*Fig. 9*): it inverts the mucous edges completely and includes all the vessels, being, in my opinion, preferable to the Connell, about whose efficacy for preventing leakage and controlling hæmorrhage I have always entertained the gravest doubts. My own modification is to use two needles, so that the suture line shall not finish at a corner—the weak spot of any anastomosis. The suture having reached the right-hand corner, and being tied to the stay stitch, the needle is taken straight out at the corner of the opening in the transverse colon, straight in at that on the cæcum, and

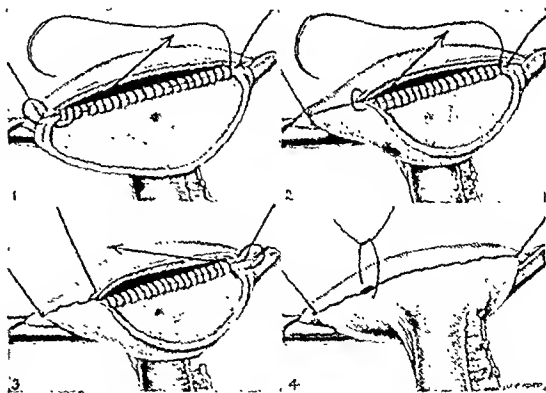


FIG. 9.—Detail of return layer of hæmostatic suture.

The suture having reached the right-hand corner, and being tied to the stay stitch, the needle is taken straight out at the corner of the opening in the transverse colon, straight in at that on the cæcum, and

pulled tight on the mucosa (*Fig. 9, 1*): it is again passed from within out through the wall of the transverse colon  $\frac{1}{2}$  in. beyond the corner, from without in through the cut edge of the cæcum the same distance beyond the corner, and pulled tight. Two more double stitches are taken and pulled tight on the mucosa, so that the first half-inch of the return layer is now inverted at the right-hand end (*Fig. 9, 2*). The needle is then passed from within out through the edge of the transverse colon just beyond the last stitich, and laid aside. The second needle is used to invert the left-hand corner in a similar manner (*Fig. 9, 3*). It is first passed straight out through the cæcal corner and straight in through that in the transverse colon, and pulled tight on the mucosa. The suture is continued from left to right, each stitich, as it is pulled tight from within, inverting the two edges, till the other end of the suture is reached. The needle is then passed through the cæcal wall from within outwards. The two ends, both of which appear on the outer coat, are now tied, inverting the last bit of the edge (*Fig. 9, 4*).

The suture which was left on the stay suture fixing the outer coat of the cæcum to the longitudinal band on the transverse colon at the left-hand end is now taken, and a continuous Lembert suture is put in from left to right, inverting the line of the hæmostatic suture; this is tied to the stay suture left at the right-hand end. A third layer, a running Lembert suture inverting the edges still further, is then inserted all round the anastomosis, which is turned round to enable its posterior surface to be reached.

The raw areas in the abdomen are now covered with peritoneum (*Fig. 10*). The gap between the upper leaf of the mesentery of the ileum and the cut edge of the transverse mesocolon has been folded on itself, and is closed by a short continuous suture. The lower leaf of the mesentery is similarly sewn to the edge of the parietal peritoneum, where it was divided on the outer side of the ascending colon. The blind end of the transverse colon is laid on the bed of the hepatic flexure, and fixed to the edges of the peritoneum on the posterior abdominal wall by a few interrupted sutures. The cut edge of the omentum, if redundant, is fixed by one or two stitches over the anastomosis.

The whole area of operation is lightly mopped with flavine solution, and the abdomen closed.



FIG. 10.—The operation of ileocecal transplantation completed.

## DISCUSSION.

The operation here described is difficult and severe, and by no means without risk. It is, however, so essentially sound in principle, and has been so successful in the two instances recorded below, that I intend to perform



it in further cases where the disability justifies a procedure of such magnitude. One difficulty is that the patients are wasted, toxie, and discouraged, and already in poor condition to withstand a severe ordeal. Extensive adhesions from previous operations must be separated, a time-consuming task, before resection can be commenced.

In the operation itself the chief difficulty is that of making a secure and leak-proof transplant into the side of the colon. Care is required in cutting the cæcal patch to ensure that its size is conformable to that of the opening in the colon, and that the ileal opening is at its centre and not near enough to any edge to suffer damage to its musculature or nerve-supply. The suture of the posterior cæcal wall to the transverse colon is the least secure part of

the anastomosis, since the whole of this wall presents a rather indefinite surface uncovered by peritoneum, an enlarged 'danger area'. For this reason I have used interrupted stitches for the posterior half of the first layer, and have reinforced the whole anastomosis with a third layer of sutures.

After operation there remains the further difficulty of getting the bowels open without putting any strain on the new stoma. To minimize this strain, I have had the patients in hospital for a week before operation, and given them 4 oz. of Petrolagar daily. On the morning of the operation the colon is washed out. After operation Petrolagar is again given every four hours. In spite of these precautions, it has been necessary in each case to give a small glycerin enema before the bowels were evacuated.

On account of these difficulties and dangers, I performed in a third case the



FIG. 11.—The operation of cæcal transplantation.

operation of transplantation of the cæcum. The first stages of this operation were identical with those described. An end-to-end anastomosis was then made, by the aseptic method of Pringle and Schoemaker, between the cæcum, half an inch beyond the sphincter, and a point in the transverse colon about its middle, chosen to allow the cæcum to lie naturally in the bed of the hepatic flexure after anastomosis (*Fig. 11*). I have found that the aseptic method has robbed end-to-end anastomosis in the colon of its difficulties, not because it is aseptic, but because the two ends of gut are held straight and of identical length till the suture line is complete. The junction, when finished, is a much neater piece of work than I have ever succeeded in producing by the open method. Pringle's forceps have failed me, and Schoemaker's I have found too short; those I use are made by Down Bros., and are Schoemaker's with blades 1 cm. longer than the original pattern. In this operation the result after three months is identical with that following the operation of ileocecal transplantation, but the radiograms taken three

# TRANSPLANTATION OF ILEOCÆCAL SPHINCTER 17

weeks after operation show a very definite diaphragm at the suture line, leading to marked stasis in the terminal ileum.

The operation of transplantation of the cæcum is shorter, easier, and probably safer, than that of transplantation of the ileocæcal valve. When resection of the ascending colon is required for non-malignant conditions in which the cæcum is undamaged, as in the case reported, it is probably the better; but when, as in most cases following previous operations, the cæcum is damaged, the operation of transplanting the valve alone into a new cæcum made from colon which is still healthy, is more likely to give results that are permanently satisfactory.

## CASE REPORTS.

*Case 1.*—Female, age 37. Transplantation of the ileocæcal sphincter.

**HISTORY.**—Appendicitis in 1922; typhoid fever in 1924; tonsillectomy in 1927. Laparotomy in 1928 for pyrexia with pelvic symptoms, supposed to be due to salpingitis; only a small cyst of the right ovary was found, and this was removed. Since the last operation, the patient had never been well, and had to abandon her

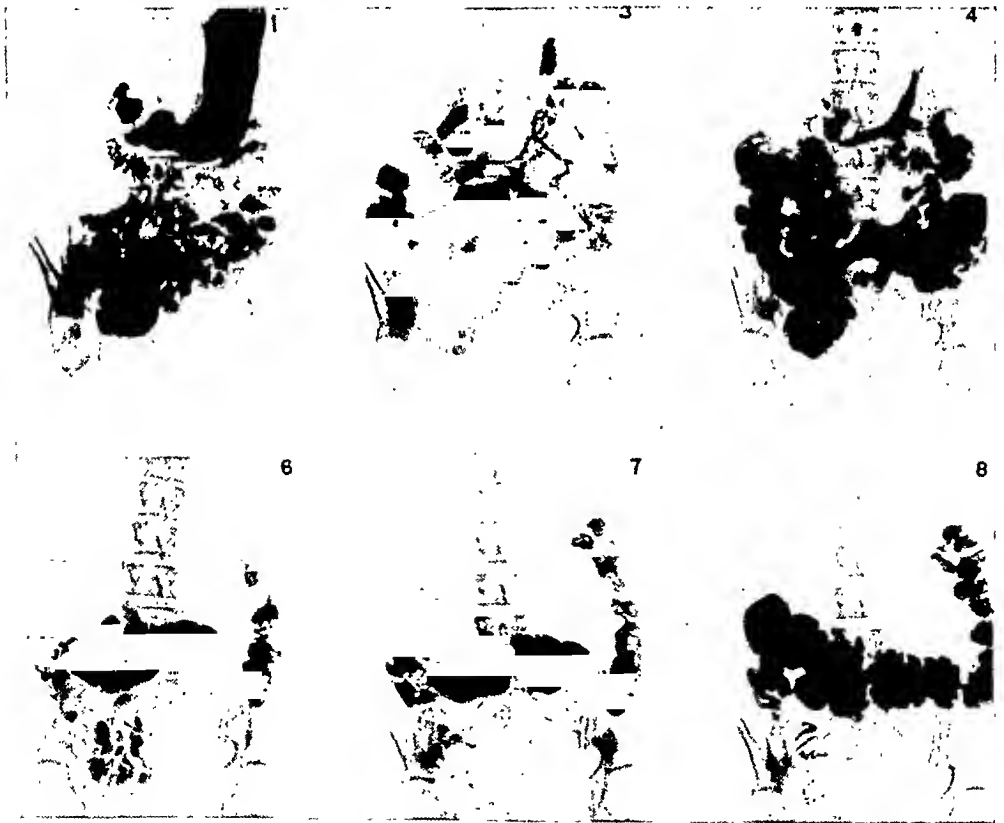


FIG. 12.—*Case 1.* Barium meal three weeks after operation.

work. She complained of a constant dull ache in the right flank and iliac fossa, increased by fatigue, and sometimes very severe. She always had an evening temperature.

ON EXAMINATION.—The patient was admitted to New Lodge Clinic for investigation. While under observation her temperature rose every night to between  $99^{\circ}$  and  $99.8^{\circ}$ . The leucocyte count before admission showed 12,000 white corpuscles per c.mm., with 74 per cent polymorphonuclear cells: in the clinic three counts showed a less marked, but definite, leucocytosis, with a relative increase in polymorphonuclear cells. The red-cell count on admission was 3,700,000 cells per c.mm., and the hæmoglobin percentage 60. After daily injection of iron and arsenic for three weeks, the red count only rose to 4,080,000 cells per c.mm., and the hæmoglobin percentage to 66.

A fractional test-meal showed a marked deficiency in free hydrochloric acid.

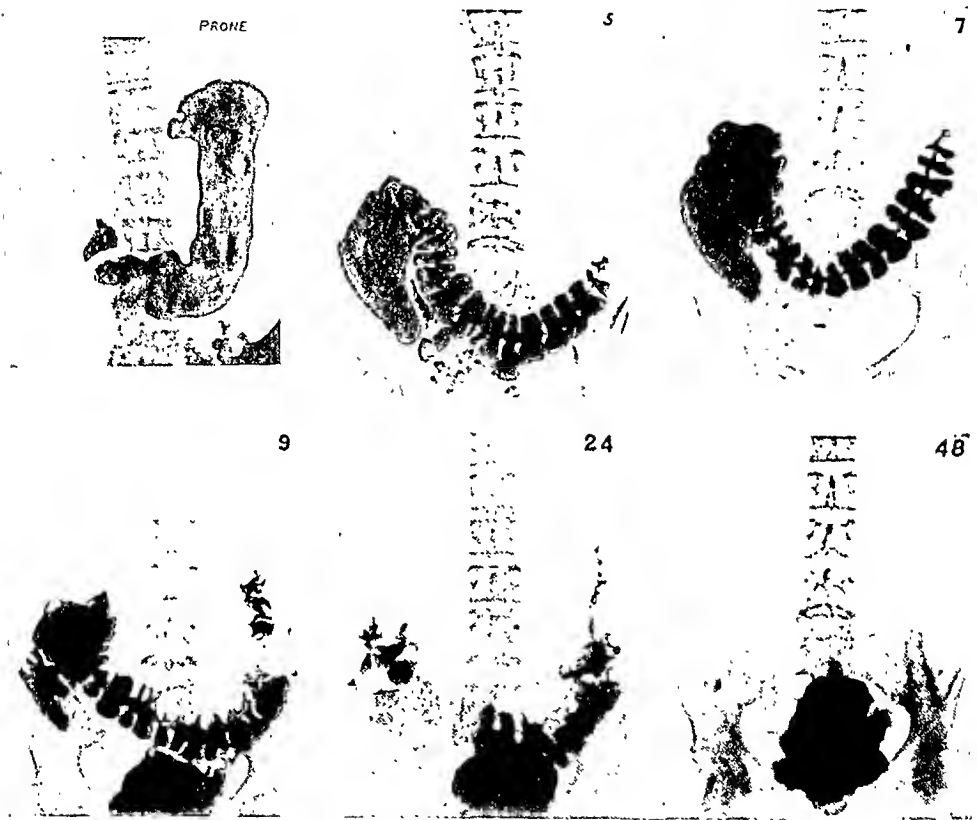


FIG. 13.—Case 2. Barium meal before operation.

A barium meal showed no abnormality in the stomach or duodenum; there was no abnormality in the outline of the large or small intestine, and no delay. By palpation under the fluorescent screen when the colon had been made visible, it appeared that the tenderness was confined to the cæcum and ascending colon; the cæcum, which just dipped into the true pelvis, was less tender than the ascending colon.

Repeated examination of the stools disclosed no abnormality. Cultures gave *Bacillus coli communis* and enterococci in normal numbers; no other organisms were found. A prolonged search failed to reveal amœbic cysts or tubercle bacilli. There was no excess of food residue, no excess of mucus or epithelial cells, no pus cells, and no blood, either obvious or occult.

A catheter specimen of urine showed no abnormality, and cultures remained sterile. Examination by a gynaecologist failed to reveal any cause in the pelvis to account for the pain or the pyrexia.

## TRANSPLANTATION OF ILEOCÆCAL SPHINCTER 19

A diagnosis of chronic infection of the ascending colon was made. The patient was kept in bed on a non-residue diet, and her bowels regulated by  $\frac{1}{2}$  oz. of paraffin three times a day. A drachm of dilute hydrochloric acid was also given three times daily. In addition she was given *acidophilus* culture, chalkaolin, bismuth salicylate, and belladonna. She was also given diathermy to the right side of the abdomen. No improvement resulted from this treatment.

OPERATION (May 30, 1930).—Spinocain, followed later by ether. Resection of ascending and one-third of transverse colon: transplantation of ileocæcal sphincter into transverse colon.



FIG. 14.—Case 2. Barium meal three weeks after operation.

REPORT ON SPECIMEN REMOVED (Dr. G. W. Rake).—Three piece of large intestine. The largest portion includes the whole circumference of the wall of the colon and measures 7 cm. in length. The longitudinal muscle bands are firmly contracted so that they are readily visible and palpable as raised white firm strands. The serous surface is thick, white, and opaque. There are several small adhesions, and one larger one which has been ligatured. The subserous adipose tissue is about normal in amount. In one place is a subserous hæmatoma the size of a cherry-stone, apparently resulting from the surgical procedure since it lies in close relation to a catgut suture. The muscular coat is not thickened and appears normal; the submucosa is somewhat thickened. The mucosa is free from all sign of inflammatory change or ulceration. The lymphoid follicles are normal in size and number. The mucosa shows a general dark-brown colour with irregular areas of a darker grey pigmentation; it is not thickened.

The two smaller portions have the same general appearance as the larger. Microscopical examination shows a very advanced degree of pigmentation in the mucosa. The pigment, which is in the form of coarser and finer, golden-brown, melanin-like granules, lies mostly within large mononuclear cells, although part is apparently free in the supporting stroma of the mucosa. It is most abundant just within the muscularis mucosæ, but extends up, in decreasing amount, between the glands to the surface. Epithelium and lymph follicles remain free from the pigment. The mucosa is otherwise normal. The submucosa is slightly thickened, with increase of fibrous tissue. The vessels show a slight degree of endarteritis. The muscle coats

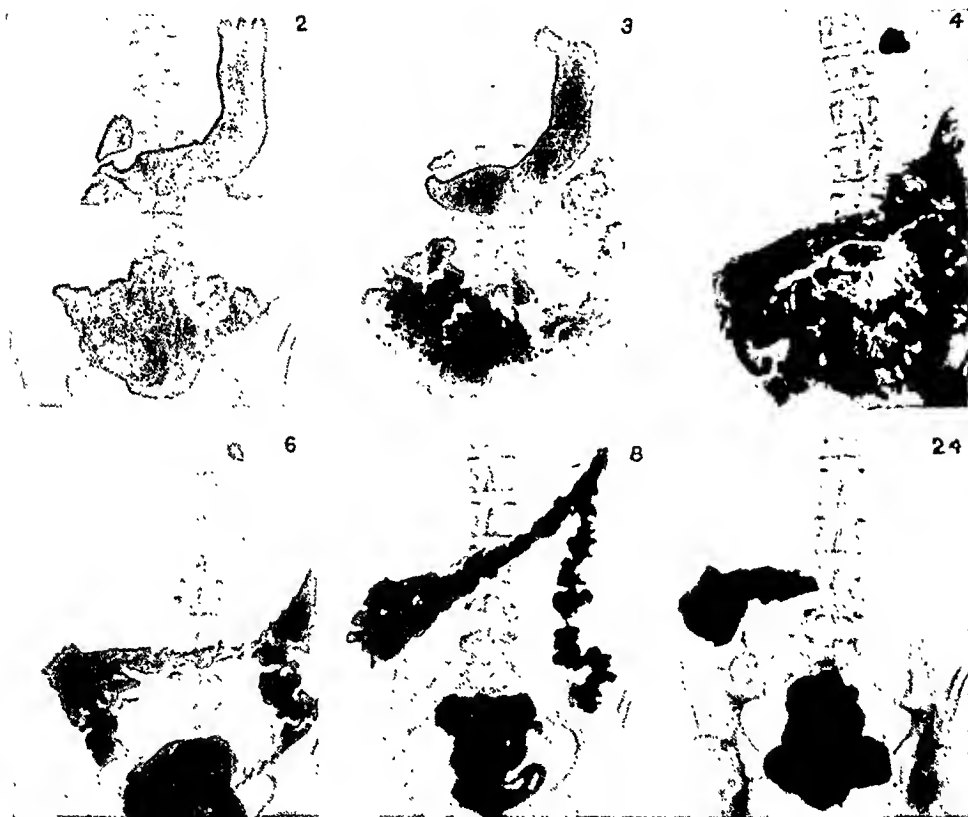


FIG. 15.—Case 2. Barium meal four months after operation. In all three series of this case a developmental pouch can be seen near the cardia.

appear normal; the plexus shows no abnormality. The subserosa is slightly thickened and fibrous. In one localized area in the muscle coat, and, to a lesser degree, in the submucosa, the capillaries are surrounded by a zone of polymorpho-neutrophil and eosinophil leucocytes together with monocytes. This is obviously of very recent origin. Its causation is not clear.

The (so-called) melanosis of the colon is of a severe degree such as is of infrequent occurrence. As far as I know such a degree of change is almost invariably associated with severe constipation.

DIAGNOSIS.—Melanosis of colon.

SUBSEQUENT PROGRESS.—

*Barium meal* (three weeks after operation—Fig. 12).—The new cæcum lies in the right hypochondrium. Barium reaches the colon in four hours, and the

# TRANSPLANTATION OF ILEOCÆCAL SPHINCTER 21

majority has left the ileum in six hours. A small residue remains in the last two inches of ileum at eight hours.

Six months after operation the patient is feeling very much better and leading an active life. Some anæmia persists and she has not yet returned to work. She has had no abdominal pain or pyrexia since the operation.

*Case 2.*—Female, age 24. Transplantation of the ileocæcal sphincter.

*HISTORY.*—Operation for gall-stones in 1923; gall-bladder and appendix removed. Second operation for adhesions in 1926. Laparotomy in 1927, right

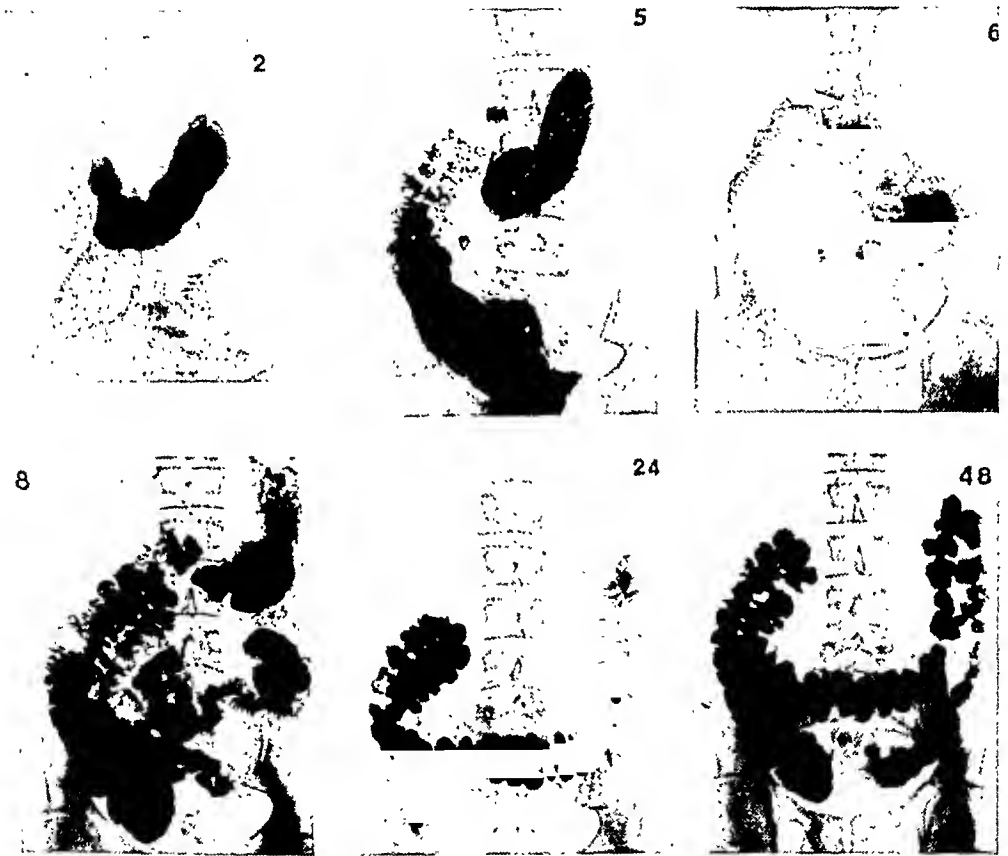


FIG. 16.—*Case 3.* Barium meal before operation.

ovary removed. The patient was admitted to New Lodge Clinic in 1928 for pain in the gall-bladder region; this cleared up under treatment with olive oil and tincture of hyoscyamus half an hour before meals. Shortly after this she developed a new pain low down on the right-hand side of the abdomen, over the site of the cæcum and ascending colon. The pain was constantly present, and bore no relation to the action of her bowels; it was not aggravated by meals or exercise, nor at the menstrual periods. The pain gradually got worse, and led to exhaustion, insomnia, and loss of weight. She was obliged to give up all games.

*ON EXAMINATION.*—She was readmitted to New Lodge Clinic in 1930. Marked tenderness was found over the cæcum and ascending colon, and over this area there was a good deal of muscular guarding.

A barium meal (*Fig. 13*) showed a diverticulum near the cardiac end of the stomach, causing no symptoms; there was no deformity of the ileum or cæcum,

and no stasis. When the parts were visualized it was shown that tenderness was most marked over the end of the ileum, and the caecum; there were no shadows in the right iliac fossa suggestive of tuberculous glands.

The stools contained no occult blood and no tubercle bacilli. A catheter specimen of urine showed no abnormality. The red-cell and white-cell counts were normal, and the differential white-cell count showed no abnormality. A fractional test-meal showed hyperchlorhydria and rapid emptying.

The patient was kept in bed for three months on a non-residue diet, and her bowels regulated with liquid paraffin. The pain was not improved.

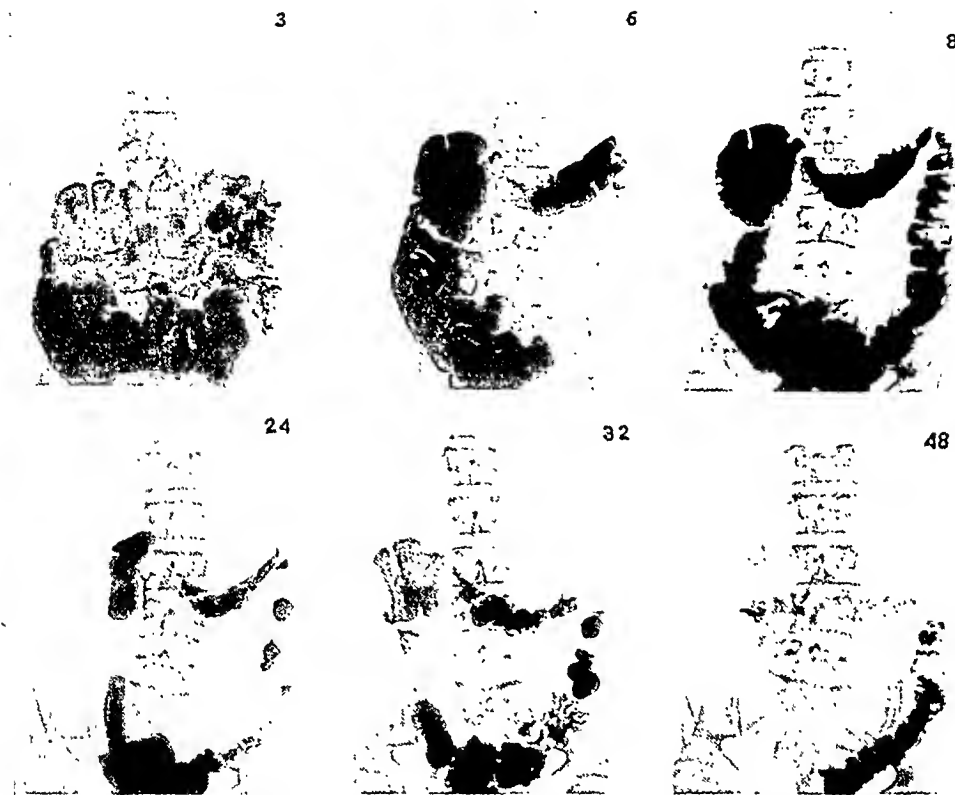


FIG. 17.—Case 3. Barium meal two and a half weeks after operation.

**OPERATION (Sept. 16, 1930).**—Laparotomy under ether anaesthesia. Very extensive adhesions were found, binding the caecum and ascending colon to the parietal peritoneum of the right iliac fossa and anterior abdominal wall. The caecum, ascending colon, and half of the transverse colon were removed, and the ileocaecal sphincter was transplanted laterally into the transverse colon 2 in. beyond its blind end.

**SUBSEQUENT PROGRESS.**—

**Barium Meal (three weeks after operation—Fig. 14).**—There is a certain amount of delay in the passage of the meal at all stages: some residue remains in the stomach at five hours, and a considerable amount occupies the lower coils of the ileum at eight hours. The new caecum lies in the right flank at the level of the iliac crest: the transverse colon runs an almost straight course.

**Barium Meal (four months after operation—Fig. 15).**—The passage of the meal through the alimentary canal is normal. All the barium has left the small intestine in seven hours: a residue remains in the new caecum at twenty-four hours.

## TRANSPLANTATION OF ILEOCÆCAL SPHINCTER 23

Four months after operation the patient is feeling well, is leading an active life, and has put on 10 lb. in weight. There is a little tenderness on deep pressure in the right iliac fossa.

*Case 3.*—Male, age 39. Transplantation of cæcum.

**HISTORY.**—Complained of recurring attacks of severe abdominal pain, from which he had suffered for twenty years. He had these attacks every three or four months, and each attack was identical and lasted about ten hours. He was always constipated during an attack. Pain commenced with a dull ache in the gall-bladder



FIG. 18.—*Case 3.* Barium meal three months after operation.

region, which gradually got worse, till it was very severe. At the height of the pain he vomited, but without any relief. After an enema the bowels were opened, and the pain rapidly subsided, being nearly gone in three hours, but leaving a soreness behind for the whole of the next day. In 1925 laparotomy was performed and a peritoneal band running from the gall-bladder to the duodenum was divided: the appendix was also removed. After this operation he remained free from pain for more than a year, but since that time the attacks have recurred as before. Latterly he has passed blood per rectum during an attack.

**ON EXAMINATION.**—No abnormality was discovered on physical examination, beyond the scar of the old laparotomy, and that of a previous herniotomy. A barium meal (*Fig. 16*) showed the stomach, duodenum, and small intestine to be normal. The transverse colon hangs down in a pronounced loop, its proximal third running vertically downwards parallel to the ascending colon, so that the hepatic flexure



is sharply angulated. The majority of the meal is still in the colon at forty-eight hours, the cæcum still being full. There is no evidence of localized obstruction.

OPERATION (June 16, 1930).—Resection of ascending, and half of transverse colon : anastomosis of cæcum to transverse colon.

SUBSEQUENT PROGRESS.—

*Barium Meal* (two and a half weeks after operation—*Fig. 17*).—The cæcum lies in the right hypochondrium and is markedly distended, being separated from the transverse colon by a narrowed segment. The transverse colon now lies almost horizontally. Barium remains in the lower ileum up to twenty-four hours, and in the cæcum up to thirty-two hours.

*Barium Meal* (three months after operation—*Fig. 18*).—The diaphragm at the cæco-transverse junction has disappeared. Passage of barium along the alimentary canal is normal.

Six months after operation the patient has had no further attacks of pain, and has returned to work.

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## CHRONIC FOLLICULAR GASTRITIS: WITH A REPORT OF NINE CASES.\*

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ALTHOUGH it has long been the custom to associate chronic ulcer of the stomach and duodenum with persisting indigestion accompanied by periodic pain, vomiting, hæmorrhage, retention, and changes in the chemical content of the gastric juice, it is now definitely established that these signs and symptoms may occur in the absence of ulcer. It has been proved that chronic gastritis (simple non-specific inflammatory disease of the stomach wall) can itself be responsible for the complete 'pyloric syndrome'.

The studies of Konjetzny, Nicolaysen, and others in the last few years have directed attention to one particular form of chronic inflammation of the stomach wall, under the name of 'chronic follicular gastritis'. It is the purpose of this communication to review certain aspects of the subject of gastritis, and to place on record nine clinical cases of the chronic follicular type of the disease. In this series chronic indigestion and hæmorrhages were the prominent clinical features. The cases were all treated by partial gastrectomy, and in the gross specimens no ulcers were found in the portions of stomach removed at operation. There is no reason to believe that ulcers may have been overlooked at the time of resection. In every case there was found in the stomach wall a characteristic inflammatory change exhibiting the gross and microscopic features of 'chronic follicular gastritis'.

### HISTORICAL.

About a century ago gastritis began to be mentioned in medical writings. Broussais is credited with the first description of the lesion. It is quite possible, however, that in the literature of that period post-mortem auto-digestion was at times mistaken for a true organic lesion, and 'gastritis' perhaps appears in the autopsy records more often than it should were such a possibility ruled out. Fixation of the stomach immediately after death by the intraperitoneal injection of formalin served to establish the comparative rarity of true gastritis, but the authority of tradition was so strong that the error persisted in medical writings. Towards the end of last century the cellular pathology of the body organs was rapidly developed and correlated with the clinical signs and symptoms of disease, but post-mortem autodigestion

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excluded the stomach from satisfactory and convenient study until radical surgical procedures provided fresh material which could be placed in fixatives in the operating-room immediately after removal from the body. Up to twenty years ago only a few of the pathologists could speak with authority on the histopathology of the stomach, but during the last decade the surgeons have provided abundant material for the detailed study of the gross and microscopic appearance of the organ in various diseased states.

One outstanding result of these studies has been to bring to the fore the subject of gastritis. The proposal to group the cases showing the chronic follicular lesions as a separate type of gastritis has followed the recognition of its characteristic histopathology, associated with a particular type of clinical history, and a fairly constant response to radical surgical treatment by partial gastrectomy.

Hunger pains, hyperchlorhydria, and delayed emptying of the stomach formed the 'pyloric syndrome' of Soupault (1901). This group of symptoms, through the work of Moynihan and others, came to be definitely associated with chronic ulcer of the pyloric region. If vomiting and hæmorrhage were also present, a diagnosis of ulcer on clinical grounds alone was quite justified. It was not known that the pyloric syndrome could be present without ulcer until Konjetzny and other writers on this subject published their observations during the last few years.

Excluding syphilis, tuberculosis, and plastic linitis, Ewald distinguished three forms of gastritis: (1) Simple; (2) Mucous, or myxorrhœa gastrica; (3) Atrophic. The pathological changes which accompanied these three forms were numerous and varied. In chronic simple and in mucous gastritis the stomach was described as being usually large, and the mucous membrane pale and covered with tenacious mucus. Ecchymosis, pigmentation, and hæmorrhagic erosions were seen, and the surface was at times mammillated (gastritis polyposa). Parenchymatous degeneration and interstitial inflammation were seen under the microscope, especially in the pyloric region. In the advanced atrophic type the mucous membrane exhibited complete atrophy (pangastritis), and the muscularis mucosæ was hypertrophied. A generalized hardening and shrinking of the whole stomach was known to occur in late cases (cirrhosis of the stomach). Perigastritis (gastritis adhesiva) with organized adhesions to the surrounding structures was a recognized accompaniment of gastritis.

This summary of the conventional conception of gastritis does not include the clinical feature of *bleeding from the stomach*, or the histological finding of *lymph follicles with germinal centres* in the mucous membrane. Hale White described twenty-nine cases of hæmorrhage from the stomach without lesion of the mucosa under the name of *gastrostaxis*, and many other surgeons have observed blood oozing from multiple points of the apparently intact gastric lining. An association with gastritis was not suspected, and no histological reports on the cases are available. Lymph follicles with germinal centres are infrequently mentioned in the earlier descriptions of gastritis except in the writings of Stoerk and in those of a few other accurate observers.

A few years ago, under the influence of Einhorn and other clinical workers, the term *gastritis* came to take on a functional meaning instead of a true organic significance. It has been, and still is, used by many to indicate a neurosis, or the result of a neurosis. To avoid confusion it is used in this paper to denote a structural change in the stomach wall with the characteristics of chronic inflammation.

In 1913 Heyrowsky made a histological study of fragments of gastric wall which he had removed from the stomach during the operation of gastroenterostomy performed for the relief of retention in ulcer or cancer cases. In none of the specimens was normal mucosa found. An inflammatory change was constantly present, as evidenced by interstitial exudation and hyperplastic changes in the epithelium. When the technique of partial gastrectomy was brought to its present satisfactory state, studies were made of the part of the stomach removed for cancer or ulcer. These specimens always exhibited a gastritis associated with the primary lesion.

The next step in the development of the subject was the suggestion made by several surgeons, of whom Finsterer was among the first, that a partial gastrectomy should be carried out in those cases where the clinical history pointed emphatically to the presence of organic disease, but where no ulcer or cancer could be found at operation. This proposal was brought forward because several surgeons had performed successful and beneficial partial gastrectomies in cases where an external inspection of the stomach suggested that a chronic ulcer existed but an intact mucous membrane was found in the specimen after removal.

Konjetzny, in 1924, gave a detailed description of eight cases in which resection had been carried out in the absence of ulcer. The patients had all suffered from some variety of indigestion. Hæmorrhage was a frequent complication. Study of the specimens revealed no ulcer, but definite gross and microscopic evidence of disease, and his work led to the recognition of *chronic follicular gastritis* (the name was suggested by Dobrovolski) as a clinical and pathological entity. Reports of other cases soon followed, and it is now established that the pyloric syndrome can be present without ulcer. Faber goes so far as to state that the pyloric syndrome can no longer be considered to be caused by ulcer, but by juxtapyloric gastritis. He believes that the symptoms and signs constituting this syndrome are only to be interpreted as evidence of disease in the pyloric region.

**Definition.**—By ‘chronic follicular gastritis’, then, is meant an inflammatory change in the stomach wall of long duration, with variable gross characteristics but constant microscopic appearances, and a clinical course marked by dyspepsia and often by hæmorrhage. The histological picture is that of inflammatory exudation into the mucosa. The exudate consists of eosinophils, plasma cells, and many lymphocytes, which are aggregated in places to form large lymph follicles with germinal centres. The lesion is probably related to the follicular form of duodenitis, but for the purpose of the present discussion is considered a separate lesion. The etiology of chronic follicular gastritis, and the relation between it and chronic gastric ulcer, are still unsolved problems.

## CASE REPORTS.

The microscopic study of the sections from the following cases is based upon tissue which was fixed at the operation, or soon afterwards, embedded in paraffin, and the sections stained with differential stains.

**Case 1.**—Miss I. G., age 56, housekeeper. Admitted April 19, 1928, complaining of belching of gas.

**HISTORY.**—At the age of 15 she was first troubled with epigastric pain, accompanied by flatulence, coming on about two hours after meals. The attacks would last about a month at a time, and occurred about twice yearly. Ten years ago this indigestion ceased, and she was free from symptoms for five years. She then began to complain of eructation of gas, with no periodicity or relation to the taking of food. No hæmorrhage. Nutrition maintained.

**ON EXAMINATION.**—Test-meal gave a small quantity of gastric content with low acidity (4·6 to 8), traces of blood, no free HCl. No blood in the stool. X-ray examination showed an interruption of peristaltic waves in the pyloric region, with a filling defect, and six-hour retention.

**CLINICAL DIAGNOSIS.**—Carcinoma engrafted upon ulcer.

**OPERATION** (May 3—Dr. W. L. Barlow).—Stellate scarring noted on the anterior surface of the pylorus. There was also a slight hour-glass deformity due to thickening and shortening of the gastro-hepatic omentum. An enlarged lymph gland was noted in the gastrocolic omentum. There were adhesions in the lesser peritoneal sac between the stomach and pancreas. Partial gastrectomy (Polya method).

**PATHOLOGICAL REPORT** (S-28-566).—The specimen consists of a portion of stomach 12 cm. in length and 4·5 cm. in width before opening. The inner surface shows no evidence of any ulceration.

**Microscopic Appearances.**—In many areas the mucous membrane is so intensely infiltrated with inflammatory cells (plasma, round cells, and eosinophils) with large germinal centres, that the gastric glands cannot be recognized. Dilated blood-vessels appear in large numbers in the mucosa and submucosa. There is extreme thickening of the muscularis mucosæ. The submucous area is entirely replaced by fibrous tissue. There are many large blood-vessels with thickened walls in the subserosa. Follicular gastritis.

**SUBSEQUENT COURSE.**—Twenty-nine months after operation (October, 1930), general health excellent; no indigestion. Works as a domestic servant.

**Comment.**—The X-ray findings pointed to organic disease, and deformity of the stomach was found at operation, but microscopic study of the specimen showed no ulcer or cancer. A typical picture of chronic follicular gastritis was found.

**Case 2.**—Mr. C. S., age 34, clerk. Admitted Dec. 18, 1921.

**HISTORY.**—Indigestion for six months: at first a feeling of fullness, then eructations of acid-tasting material. One week previously he felt a sudden heavy weakening pain in the upper abdomen, which usually came on one or two hours after food. Slightly later the pain would appear about one hour before meals and be relieved by eating. On the day of admission there was a sudden stabbing pain in the upper abdomen, spreading to the back and right shoulder.

**ON EXAMINATION.**—There was a general rigidity over the right upper quadrant of the abdomen.

**FIRST OPERATION** (Dec. 18—Dr. A. Hutchison).—A perforation was seen on the anterior wall of the duodenum, with free escape of fluid; this was closed. Posterior gastro-enterostomy. The patient developed pleurisy with effusion six days after operation, but made a good recovery, and was discharged 'well' on Jan. 5, 1922.

**RE-ADMISSION** (Oct. 28, 1928).—Almost seven years after operation the patient consulted his physician on account of tarry stools. The following day the stool

contained dark blood, and he experienced a feeling of profound weakness. There was a severe secondary anaemia (Hb 55 per cent); a barium meal showed a functioning gastro-enterostomy, but no evidence of gastric ulcer, cancer, or marginal or jejunal ulcer.

**SECOND OPERATION** (Nov. 13—Dr. W. L. Barlow).—Numerous adhesions divided between transverse colon, omentum, and anterior abdominal wall, but no adhesions noted in the region of the duodenum. No external evidence of ulcer. A pouch-like projection on the lower surface of the first part of the duodenum indicated the site of the previous ulcer. Thickening of the pyloric ring was noted. The epiploic foramen was obstructed. Pylorectomy, with removal of the first part of the duodenum.

**PATHOLOGICAL REPORT** (S-28-1369).—The specimen consists of a portion of stomach and duodenum, with a few adhesions in the region of the pylorus. Pyloric ring thickened. There are about half a dozen areas of hæmorrhage in the mucosa of the stomach; but no ulceration, acute or old, can be made out.

**Microscopic Appearances.**—Lymph follicles with germinal centres are present in the mucosa. The gland tubules are separated by round cells, plasma cells, and eosinophils. At the pylorus there is a diffuse fibrosis of the deepest layers of the mucous membrane, and there are very few gland tubules or acini to be seen. There is a pronounced follicular duodenitis.

**DIAGNOSIS.**—Chronic follicular gastritis and duodenitis.

**SUBSEQUENT COURSE.**—Twenty-three months after operation (October, 1930), excellent general health, no indigestion, no hæmorrhages. Cured.

**Comment.**—The interest in this case centres around the sudden hæmorrhages without indigestion which came on just before the second admission to hospital.

**Case 3.**—Mr. R. B., age 37. Admitted Oct. 7, 1928.

**HISTORY.**—There was a history of a gnawing burning epigastric pain of ten years' duration. At times a dull aching pain was felt in the chest. The pains originally came on at rare intervals, but they had been steadily increasing in frequency and duration. Vomiting frequently after solid food; no bleeding. Obstinate constipation. Pains were relieved by taking water or soda, and aggravated by food. Loss of 40 lb. weight in a few months.

**ON EXAMINATION.**—First barium series, negative. Second barium series showed evidence of a duodenal ulcer with 50 per cent retention at six hours. Test-meal, 75 c.c.; total acidity 78 per cent, free HCl 56 per cent. No occult blood in gastric contents or stool.

Sippy régime begun. Relief of all pain in twenty-four hours; vomiting stopped; hæmoglobin rose from 78 to 105 per cent. Discharged Nov. 7, feeling well except for a sensation of weight in the epigastrium.

**RE-ADMISSION** (Nov. 17).—The patient complained of a return of vomiting and a distressing feeling of weight in the region of the heart.

**OPERATION** (Nov. 24—Dr. C. K. P. Henry).—Removal of atrophic kinked appendix. Stomach and duodenum normal on inspection. In view of history and X-ray findings, partial gastrectomy (Polyá).

**PATHOLOGICAL REPORT** (S-28-1416).—The specimen consists of a portion of the pyloric end of the stomach. The mucosa is covered with tenacious mucus. It is pale, with a few small patches of petechial hæmorrhage. The pyloric ring is not thickened; there is no ulcer.

**Microscopic Appearances.**—Mucosa shows abundant follicles which are placed close to the muscularis mucosæ. Very little infiltration of the mucosa. No thickening of the muscularis mucosæ. In the muscular coat there are many collections of inflammatory cells (polymorphs) arranged about small blood-vessels. Collections of plasma cells, round cells, and eosinophils are also found in the subserous layer.

**DIAGNOSIS.**—Chronic follicular gastritis.

**SUBSEQUENT COURSE.**—Twenty-three months after operation (October, 1930), no pain or vomiting; gain in weight 37 lb. Working as upholsterer. Very slight epigastric discomfort after indiscretions in diet or heavy exertion. Cured.

**Comment.**—A thorough trial of conservative treatment failed to control the symptoms. The microscopic examination of the stomach revealed surprisingly little change in the mucosa, when the duration and the severity of the symptoms were considered. Collections of inflammatory cells were found in the muscularis and subserosa.

**Case 4.**—Mr. J. P., age 40, labourer. Admitted Nov. 19, 1928, complaining of abdominal pain and vomiting.

**HISTORY.**—Nine months before admission he suffered an attack of abdominal pain with vomiting lasting one week. Since that time had complained of epigastric pain, anorexia, and vomiting. There were alternate periods of diarrhoea and constipation. The pain was unaffected by the taking of food. Considerable loss of weight.

**ON EXAMINATION.**—Slight splinting of the abdominal muscles in the epigastrium and right upper quadrant was revealed, and there was definite tenderness on pressure over the upper part of the right rectus muscle. X-ray negative.

**OPERATION** (Oct. 26—Dr. C. K. P. Henry).—The duodenum was covered by strands of fine vascularized adhesions. In view of the clinical history a partial gastrectomy (Polyú) was performed. Patient discharged nineteen days later; no complaints.

**PATHOLOGICAL REPORT** (S-29-1425).—The mucous membrane shows infiltration with wandering cells (plasma cells, round cells, eosinophils), intense in some areas. There are many follicles with germinal centres occupying the intermediate and deepest layers of the mucosa. The muscularis mucosæ is much thickened, and, in places, split into layers. There is a mild follicular duodenitis.

**DIAGNOSIS.**—Chronic follicular gastritis.

**SUBSEQUENT COURSE.**—Working as a labourer (October, 1930); very rough food; very good health; free from indigestion. Cured.

**Comment.**—The clinical history of previous acute illness suggested penetrating ulcer. At operation the stomach and duodenum appeared normal. A resection was carried out on account of the definite clinical history.

**Case 5.**—Mrs. A. M., age 47. Admitted Feb. 4, 1929.

**HISTORY.**—The patient complained of recurring attacks of abdominal pain, accompanied by vomiting of large quantities of watery brown material. The last attack was particularly severe and occurred on the day of admission.

**ON EXAMINATION.**—The abdominal muscles were splinted, and there was an acutely tender area in the epigastrium, just to the left of the mid-line. Pulse 80, temperature 99.4°.

**DIAGNOSIS.**—Penetrating peptic ulcer.

**OPERATION** (Feb. 4—Dr. A. Stewart).—Gastro-hepatic omentum adherent to the anterior abdominal wall. This was separated and it was found that the same portion of omentum was firmly adherent to the lesser curvature of the stomach about midway between the pylorus and cardia. The gastric wall was thickened and œdematous at this point. Diagnosis: perforating gastric ulcer sealed by omentum. Partial gastrectomy, 'Balfour' method, with removal of the supposed ulcer.

**PATHOLOGICAL REPORT** (S-29-134).—No ulcer seen.

**Microscopic Appearances.**—The mucous membrane is packed with inflammatory cells (round cells, plasma cells, eosinophils). There are many follicles with germinal centres. The follicles are very large, and reach almost to the surface. There are many crypts or defects on the mucous surface leading down to the follicles or their neighbourhood. There is an irregular thickening of the muscularis mucosæ. In the subserosa there is œdema and infiltration with inflammatory exudate. One cyst seen.

**DIAGNOSIS.**—Follicular gastritis.

**SUBSEQUENT COURSE.**—A period of severe vomiting followed the operation, requiring gastric lavage and intravenous salines. The patient left the hospital on

the twenty-ninth day after operation in very good condition. She is entirely free from digestive complaints eighteen months after operation.

**Comment.**—In this case the acute onset and the severity of the symptoms suggested penetrating ulcer with peritonitis. No ulcer could be found in the specimen.

*Case 6.*—Mr. H. C., age 32, mechanic. Admitted June 20, 1928.

**HISTORY.**—Had suffered for four years with dull aching abdominal pain which was continuously present day and night, but was always worse when he was hungry. Food relieved the pain for about half an hour. He brought up a mouthful of vomitus ten or twelve times a day. Blood in the stool at times. Eructations of gas. Lost 23 lb. in weight in five years. Sippy and Lenhartz diets tried without relief. Mother has stomach trouble. Father, age 65, has had two operations for a stomach ulcer. One sister is receiving treatment for gastric ulcer.

**ON EXAMINATION.**—A sharply localized point of tenderness was revealed in the epigastrium. Test-meal gave: total acidity 80, free HCl 60. Blood was detected by chemical tests of the stool. Barium series revealed a constantly deformed cap, hyperperistalsis of the stomach, and rapid advance of the meal.

**CLINICAL DIAGNOSIS.**—Duodenal ulcer.

**FIRST OPERATION** (June 25—Dr. C. K. P. Henry).—Adhesions found between the great omentum, cæcum, transverse colon, hepatic flexure, liver, gall-bladder, and anterior abdominal wall. Extensive periduodenal adhesions, and a suggestion of an ulcer on the anterior wall. Pyloric ring crushed, and ligated with silk. Posterior gastro-enterostomy. There was a slight wound infection and vomiting at intervals after the operation. Discharged on Aug. 6.

**RE-ADMISSION** (March 25, 1929).—Complained of abdominal pain which came on one or two hours after meals, relieved by the next meal. Vomiting came on fifteen to twenty minutes after each meal. The pain was often nocturnal. Test-meal gave a volume of 39 c.c., total acidity 53.5, free HCl 39.5.

**SECOND OPERATION** (April 2—Dr. C. K. P. Henry).—Adhesions previously encountered had been partly absorbed. The pyloric part of the stomach was hard and tubular. Removal of the pyloric portion of the stomach and first part of the duodenum without interfering with the gastro-enterostomy.

**PATHOLOGICAL REPORT** (S-29-382).—The specimen consists of the pyloric part of a stomach and first part of duodenum. The mucosa shows no ulceration.

**Microscopic Appearances.**—In the superficial layers of the mucous membrane the glands are separated by a slight but definite amount of cellular exudate, made up chiefly of plasma cells, but containing a few round cells and eosinophils. In the deeper layers there are large follicles with germinal centres. The pyloric musculature shows a large irregular scar (ligature).

**DIAGNOSIS.**—Chronic follicular gastritis.

**SUBSEQUENT COURSE.**—Nineteen months after operation (October, 1930) he was feeling quite well and free from indigestion.

**Comment.**—Pyloric occlusion and gastro-enterostomy failed to control the symptoms, and the patient returned in nine months for the relief of periodic pain and vomiting.

*Case 7.*—Mr. E. R., age 52, machinist. Admitted Feb. 21, 1927.

**HISTORY.**—Attacks of aching pain in the epigastrium for fifteen years. At first the pain came on two or three hours after eating, and was relieved by food. For the past few years the pain had no relation to the taking of food. The attacks came on four or five times yearly, with long periods of relief. Attacks were always preceded by diarrhoea. No bleeding.

**ON EXAMINATION.**—Test-meal: 25 c.c., total acidity 15, free HCl 1. Occult blood present. Discharged on medical treatment.



**RE-ADMISSION** (Jan. 20, 1929).—Complaining of abdominal pain, loss of weight, weakness, anorexia. Slight anæmia (Hb 72 per cent). Occult blood detected in stool. Test-meal gave 59 c.c., total acidity 12, no free HCl. Occult blood present in stomach contents. X-ray showed a small ulcer of the duodenum. He was given the Sippy régime, and left the hospital in twenty-eight days without pain.

**SECOND RE-ADMISSION** (Aug. 10).—Felt well until a month ago when there was a return of the periodic pain, which was always relieved by food. Epigastric tenderness.

**OPERATION** (Aug. 13—Dr. C. K. P. Henry).—Stomach and duodenum normal except for a small thickening of the duodenal wall. Partial gastrectomy.

**PATHOLOGICAL REPORT** (S-29-1190).—The specimen consists of a portion of a stomach measuring 7 by 4 cm. Mucosa covered with a viscid secretion. There is no ulcer.

*Microscopic Appearance.*—The mucous membrane is closely packed with large follicles with germinal centres, and the gastric glands are separated from each other by round cells, plasma cells, and eosinophils. The muscularis mucosæ is irregularly thickened and fibrosed. The submucous layer shows diffuse scarring and dilated blood-vessels. There is also a follicular duodenitis.

**DIAGNOSIS.**—Chronic follicular gastritis and duodenitis.

**SUBSEQUENT COURSE.**—The operation was followed by intractable vomiting with forty-eight hour retention. Abdomen opened, adhesions divided. Discharged Sept. 21, feeling very well. Three months after the last operation there was still gastric retention (10 per cent in six hours). Classified as improved.

**Comment.**—Prolonged conservative treatment failed to control the disease. Considerable loss of blood and anæmia were important associated conditions.

**Case 8.**—Mr. C. B., age 32, chauffeur. Admitted Nov. 5, 1929.

**HISTORY.**—Complained of attacks of colicky pain and eructations of gas, lasting from a few minutes to three hours. No relation to the taking of food. Began eleven years ago, following an attack of influenza. Periods of relief of several weeks' duration. The pain was felt along the right costal margin. Appetite good. Tarry stools. Temporary relief with soda treatment.

**ON EXAMINATION.**—Total acidity 84, free HCl 41. Gall-stones revealed by cholecystogram. Diminished sugar tolerance. Barium series showed evidence of a duodenal ulcer.

**OPERATION** (Nov. 21—Dr. E. M. Eberts).—Cholecystectomy for gall-stones. Periduodenal adhesions were noted. The external appearance of the duodenum strongly suggested the presence of an ulcer. Partial gastrectomy (Polyá) with removal of the first part of the duodenum.

**PATHOLOGICAL REPORT** (S-29-1600).—The specimen consists of the pyloric portion of the stomach and first part of the duodenum. The mucous membrane is intact. The stomach wall is somewhat œdematous. At the commencement of the duodenum there is a thickening of the wall about 1.5 cm. in length.

*Microscopic Appearance.*—The gastric mucosa is hyperæmic, and there is an interstitial exudate composed of plasma cells, lymphocytes, eosinophils, and in places a few polymorphs. There is an increase in the lymph follicles, which show large germinal centres. The muscularis mucosæ is thickened and infiltrated with inflammatory cells. In the muscular coat there are foci of inflammatory exudate. The duodenum shows a large collection of pancreatic acini scattered in the submucosa, muscularis, and subserosa. There is fibrosis of the serous coat overlying this area. There are several ducts in the pancreatic tissue. A small collection of Brunner's glands was also noted in the subserosa. A mass of smooth muscle was present in the mucosa near the pancreatic tissue.

**DIAGNOSIS.**—Chronic follicular gastritis, with misplaced pancreatic tissue in the duodenal wall. Patient discharged in twenty days in excellent general health.

RE-ADMISSION (May 9, 1930).—Complained of having had two attacks of crampy pains beginning at the level of the lower end of the abdominal incision, lasting two or three hours. X-ray showed only a normally functioning partial gastrectomy. Conservative treatment (abdominal massage) recommended.

DIAGNOSIS.—Intestinal obstruction from post-operative adhesions. Classified as improved.

**Comment.**—Of interest here is the simultaneous existence of gall-stones and gastritis, together with a pancreatic rest in the duodenum. Among the cases of gastritis treated by resection reported in the paper by Wanke there are two in which post-operative attacks of partial intestinal obstruction were noted. As in the present case, the symptoms were attributed to 'adhesions'.

*Case 9.*—Mrs. C. H., age 54. Admitted April 6, 1930.

HISTORY.—Complained of indigestion and pains in the region of the stomach. In spite of great care in the matter of diet she had suffered from attacks of indigestion all her life. The attacks would consist of abdominal distress and eructations of gas. They would last for about one week, and would then disappear completely for a few months. She had been as long as a whole year without pain. During the attacks the pain had a distinct relationship to the taking of food. Bed, rest, and Sippy régime relieved the pain for about a year.

ON EXAMINATION.—X-ray showed a deformity of the first part of the duodenum.

OPERATION (April 11—Dr. A. T. Bazin).—Dense scarring of the anterior surface of the duodenum. Dilatation of the whole duodenum. Partial gastrectomy (Polyá). Cholecystectomy.

PATHOLOGICAL REPORT (S-30-452).—The specimen consists of pyloric portion of stomach, pyloric ring, and a fragment of the duodenum. There is considerable thickening in the pyloric region. There is a broad depression in the gastric mucosa, 2 by 2.5 cm., near the pylorus.

*Microscopic Appearances.*—There is no evidence of chronic ulcer. There are many lymph follicles with germinal centres crowded in the mucosa. Irregular thickening of the muscularis mucosæ. Scars in the mucous membrane. Glands are separated by round cells, eosinophils, plasma cells, and blood capillaries. Increase in the number of capillaries in the submucous layer. The muscularis is thicker than usual. There are many distended lymphatics filled with homogeneous material in the submucosa.

SUBSEQUENT COURSE.—Six months after operation, great improvement in general health; no indigestion; no pain; no vomiting. Cured.

**Comment.**—The long duration of the disability and the slight impairment of the general health are prominent features of the case.

## CLINICAL AND PATHOLOGICAL SUMMARY OF CASES.

In the nine cases of this series the average age at operation was 43.3 years, and there were six males and three females. A family history of gastric disease was given by one patient (*Case 6*). The average duration of the symptoms was 15.8 years; the longest case had lasted 44 years, the shortest 9 months. The onset was gradual in every case excepting *Case 5*, where it was so sudden as to suggest acute perforation. Indigestion was present in all except *Case 2*, in which copious hæmorrhage was the only sign. Actual pain was noted in seven of the nine case histories; vomiting in four. The periodicity of the symptoms was a feature of several of the cases, and in

Table I.—SHOWING PRINCIPAL FEATURES OF

CASE NO.	AGE AND SEX	DURATION OF SYMPTOMS	INDIGESTION	PAIN	VOMITING	HÆMATEMESIS	OCCULT BLOOD IN STOMACH	MELÆNA	OCCULT BLOOD IN STOOL	ACIDITY
1	56 F.	Years 41	+	0	0	0	+	0	0	4/0
2*	39 M.	7½	0	0	0	0	0	+	+	
3	37 M.	10	+	+	+	0	0	0	0	78/56
4	40 M.	¾	+	+	+	0	0	0	0	
5	47 F.	6	+	+	+	0				
6*	33 M.	5	+	+	+	0	0	0	+	53/30
7	52 M.	17	+	+	0	0	+	0	+	12/0
8	32 M.	11	+	+	0	0	0	+	0	84/41
9	54 F.	44	+	+	0	0	0	0	0	

\* This patient had previously

some instances the seasonal and daily variations in relation to the taking of food were exactly those found in chronic peptic ulcer.

Loss of blood is the most arresting clinical feature. It is recorded in five of the nine case reports. Melæna, occult blood in the gastric contents, and occult blood in the stool have all been found. Hæmatemesis was not observed. The amount of blood lost by one of the patients was alarming (Case 2). One patient (Case 8) reported having had tarry stools on several occasions. Konjetzny has reported a fatal case of hæmatemesis in which gastritis (no ulcer) was found at autopsy. In this connection it is interesting

# CHRONIC FOLLICULAR GASTRITIS.

35

## NINE CASES OF CHRONIC FOLLICULAR GASTRITIS.

X-RAY FINDINGS	OPERATIVE FINDINGS	OPERATION	PATHOLOGICAL REPORT		INTERVAL SINCE OPERATION	END-RESULT
Interrupted peristalsis at pylorus Six-hour retention	Two scars. Hour-glass deformity Enlarged glands Adhesions	Partial gastrectomy (Polyá)	Follicular gastritis		Months 29	Cured October, 1930
Functioning enterostomy	Thickened pylorus Pouch in duodenal wall	Pylorectomy	Chronic gastritis and duodenitis		23	Cured October, 1930
(1) Negative (2) Duodenal ulcer with 50 per cent retention at six hours	Normal stomach and duodenum	Partial gastrectomy (Polyá)	Chronic gastritis		23	Cured October, 1930
Negative	Vascularized periduodenal adhesions	Partial gastrectomy (Polyá)	Chronic follicular gastritis		22	Cured September, 1930
	Perigastric adhesions: local thickening and œdema of stomach wall	Partial gastrectomy (Balfour)	Chronic gastritis		18	Cured August, 1930
	Thickened antrum	Pylorectomy	Chronic follicular gastritis		19	Cured October, 1930
Negative	Area of thickening in duodenal wall	Partial gastrectomy (Polyá)	Chronic follicular gastritis		14	Retention 10 per cent Improved
Gall-stones Duodenal ulcer	Periduodenal adhesions Local thickening of duodenal wall	Partial gastrectomy (Polyá) Cholecystectomy	Chronic follicular gastritis Pancreatic tissue in duodenal wall Gall-stones		6	Re-admitted for pain Diagnosis, adhesions Improved
Duodenal ulcer Duodenal diverticulum	Scarring and dilatation of duodenum	Partial gastrectomy (Polyá) Cholecystectomy	Chronic follicular gastritis Chronic cholecystitis		6	Cured October, 1930

undergone a posterior gastro-enterostomy.

to note that hæmorrhage from the urinary tract is common in granular pyelitis, where microscopic study of the diseased pelvic lining shows follicular structures (Paschkis) comparable to those of the stomach mucosa in gastritis. Excessive secretion of mucus (myxorrhœa gastrica) was not noted in this series. It is, however, well recognized as an accompaniment of chronic gastritis, and the work of Kuttner has drawn attention to the existence of two forms, one intermittent, the other continuous. Chemical examination of the gastric content after test-breakfast showed normal values in some cases, hyperacidity, hypo-acidity, or anacidity in others.

According to Zweig, the amount of diminution in acidity can be taken as an index of the degree of destruction of gastric mucosa. Hyperacidity is said by him to be present in the early stages, normal values in advanced stages, anaecidity in the final stage.

The *X-ray findings* have been inconstant. In three cases (*Cases 3, 4, and 7*) negative results were reported. In four (*Cases 1, 3, 8, and 9*) a diagnosis of ulcer was made. *Case 3* is interesting. The first barium series was negative. A second series, taken a few days later, showed radiological evidence of duodenal ulcer. At operation only follicular gastritis was found. Most of the writers on the subject report diminished gastric mobility. H. H. Berg found characteristic X-ray pictures in gastritis after the administration of a few cubic centimetres of barium mixture by mouth. A great increase in the number and size of the mucous folds was noted. G. Schwarz mentions multiple indentations of the greater curvature, and these indentations have been noted by other writers. It is stated by Zweig that an increased sensitiveness to pressure (*Druckempfindlichkeit*) over the stomach outline, with multiple indentations along the greater curvature, and an increase in the number and depth of the folds, indicate the presence of gastritis.

*Gastroscopy* has proved disappointing as an aid to diagnosis. Although the technique is not difficult, the whole of the interior of the stomach cannot be clearly seen, and—what is more important—it is extremely difficult to interpret the findings correctly. As an example, Zweig writes of: (1) Petechial or streaky reddening; (2) Excessive mucus (*catarrhus mucosus*); (3) Gastritis hypertrophicans; (4) Multiple bleeding points and small erosions (*gastritis hæmorrhagica and granulosa*); (5) Gastritis atrophicans. Although it is possible to observe all of these changes through the gastroscope, their interpretation is difficult. B. Lubarsch has described changes in the stomach in purely physiological states that appear exactly like those found in gastritis. Schüller finds that by gastroscopy one can confirm the presence of gastric cancer in every case, but although he examined many cases of proved gastric ulcer, he was able to see the lesion itself only once. In gastritis cases he was often able to observe definite changes, but found difficulty in interpreting them, and in differentiating them from the physiological changes described by Lubarsch.

*Table I* (pp. 34, 35) shows the principal features of the nine cases of chronic follicular gastritis reported above.

### PATHOLOGICAL ANATOMY.

**Gross Appearances.**—The aboral part of the stomach is usually the only portion of the organ affected. The greatest changes are found in the pylorus and pyloric antrum. The fundus is nearly always normal. Only in the advanced atrophic form of pangastritis with achylia gastrica (Faber) is the whole stomach uniformly diseased. The distribution of the lesion is not uniform, but decidedly uneven and patchy. Normal areas may be encountered interposed among portions of diseased gastric wall. Again, no two of the abnormal areas will be identical in a given case. It is indeed possible to find in different parts of one specimen, atrophy, excessive regeneration,

superficial ulcerations of the mucosa, polypoid overgrowth, and hæmorrhagic erosions.

External inspection may reveal a blush or reddening of the serous surface. Schoemaker specially emphasizes the importance of this sign, but it may be absent in true cases of gastritis. Perigastric adhesions may unite the stomach to the great omentum, the gastro-hepatic omentum, the liver, gall-bladder, colon, or anterior abdominal wall. In one recognized type the whole stomach appears to be embedded in organized adhesions (perigastritis adhesiva). The most constant gross finding is a thickening and a palpable hardening or stiffening of the stomach wall in the pyloric region. The antrum feels hard and tubular. Enlarged, soft, reddened lymph-glands are noted above and below the pylorus in about half the cases. Small diverticula are seen at times, always at the pylorus. These are generally interpreted as being the sequelæ of healed ulcers, but this is only an assumption, and one which is not always justifiable.

On opening the stomach there is seen a thick layer of slimy mucus adhering to the inner surface. No peptic ulcer can be found. Minute superficial hæmorrhagic erosions may or may not be present. When they are found they usually occupy the apices of slightly rounded projections, or they may appear as tiny slits between the folds of mucous membrane. They consist of small openings blocked by blood-clot. Superficial irregularly-shaped patches of partial destruction of the mucous membrane over circumscribed areas are often to be seen, especially in the pylorus and antrum. These areas are slightly pale, and are referred to in the literature as 'worm-eaten patches'. Active regeneration, hyperplasia, or compensatory overgrowth of the mucous membrane may lead to a papilliferous state of the mucous membrane. This process may be extremely advanced and may produce an 'état mammeloné' or gastritis polyposa. In the pyloric region this overgrowth of the mucous membrane may, by its bulk alone, become an important factor in the production of pyloric stenosis and retention. In two cases of this type Konjetzny found early cancer.

**Microscopical Appearances** (*Figs. 19-23*).—In mild cases the mucous membrane is the only layer of the gastric wall to show inflammatory change. In the advanced stages all the layers may be altered. The patchy distribution of the lesion is obvious in the histological sections. Neighbouring fields may show normal mucosa, chronic gastritis, atrophy, excessive regeneration, erosion, or mucosal ulcerations; and different parts of the same specimen will show great differences in the depth to which the disease has penetrated the stomach wall. In general, the changes are most marked at the pylorus and antrum. Seldom is the fundus involved.

The most striking, constant, and characteristic histopathological changes are to be seen in the mucous membrane. Here the tubules of the gastric glands are not in immediate contact with each other, but are separated by masses of inflammatory cells, small round cells, plasma cells, eosinophils, and connective-tissue cells. The small round cells are frequently aggregated to form follicles with large germinal centres. These lymphoid follicles are the outstanding characteristic of the lesion, and are responsible for the name 'chronic follicular gastritis'. The follicles vary in size, but are usually large

enough to occupy about half the thickness of the mucosa. Typically, they are circular or oval on section, but may be triangular or irregular in shape. The central portion, or germinal centre, is made up of rather poorly staining and fairly homogeneous cells of irregular shape, but all tending to be round or oval. About the germinal centre there is a collar of ordinary small, round or lymphoid cells.

The eosinophils of the interfollicular inflammatory cell masses are often very abundant. Accompanying the interfollicular cellular exudates the

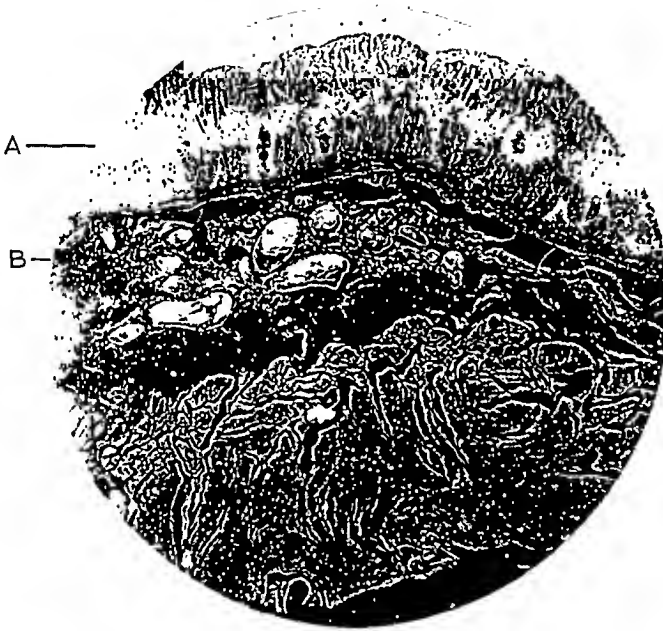


FIG. 19.—Chronic follicular gastritis, early stage. A, A row of lymph follicles with germinal centres embedded in the mucous membrane, and resting on the muscularis mucosæ, which is slightly thickened; B, Dilatation of the blood-vessels of the submucosa.

epithelial cells lining the gastric glands and their ducts show various changes. The surface epithelium, lining the actual gastric lumen, shows the most frequent alterations. These cells are normally mucus-secreting, and in sections from cases of follicular gastritis they are nearly always very active, and are filled with large masses of mucus. More advanced stages show atrophy and regressive changes, and, at times, hypertrophy or actual hyperplasia. The epithelium lining the mouths of the gastric glands is also mucus-secreting, and shows changes similar to those on the free surface.

In the normal human stomach the epithelial cells lining the elongated ducts of the gastric glands and the acini in the deepest layer of the mucosa are the true gastric secreting cells. They produce the gastric juice, and vary

in appearance in different parts of the stomach. Those in the pyloric region are simple cuboidal cells with opaque cytoplasm, and are believed to secrete a mucous material only. These pyloric gland cells are found at the pylorus itself, and in the pyloric canal and antrum. A few scattered lymphoid follicles with germinal centres are normally present in the mucosa of the antrum. Above the antrum the typical pyloric cells disappear rather suddenly, and in the body of the stomach the glands are lined by epithelial cells of three different types—namely, chief or oxyntic cells, parietal cells, and intermediary

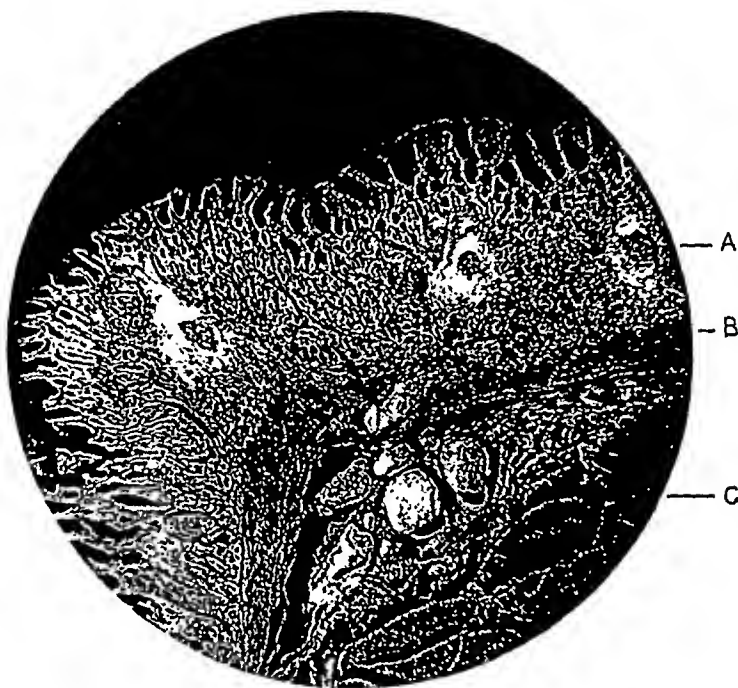


FIG. 20.—Chronic follicular gastritis. A, Three follicles with germinal centres; B, Thickening of the muscularis mucosæ; C, Dilatation of the submucous blood-vessels.

cells, the latter closely resembling the pyloric cells described above. These three varieties of cells are found throughout the greater part of the fundus and cardia, but about the cardiac orifice itself there is a narrow band of mucosa whose lining cells are morphologically similar to those of the pyloric region—that is, smaller cuboidal cells with cloudy cytoplasm (the cardiac cells).

The above description is that of the normal human stomach. In chronic follicular gastritis these specific secreting cells often show surprisingly little change, and although the tubular ducts may be separated by collections of inflammatory cells with germinal centres, the epithelial cells may appear quite normal in shape and staining reaction. Stoerk speaks of islands of glands of the intestinal type occurring in the secreting layer in gastritis cases.



Such islands have not been found in the series of cases here reported. In view of the hyperacidity so often found in gastritis it is interesting to note that neither hypertrophy nor hyperplasia of the cells secreting the gastric juice has been observed in the sections. Atrophy of the secretory epithelium is, however, a common finding. In well-established cases areas are often seen where the gland cells are practically absent, the whole mucous membrane being replaced by inflammatory tissue, cysts, and the remains of the glands and ducts, with or without follicles and germinal centres.

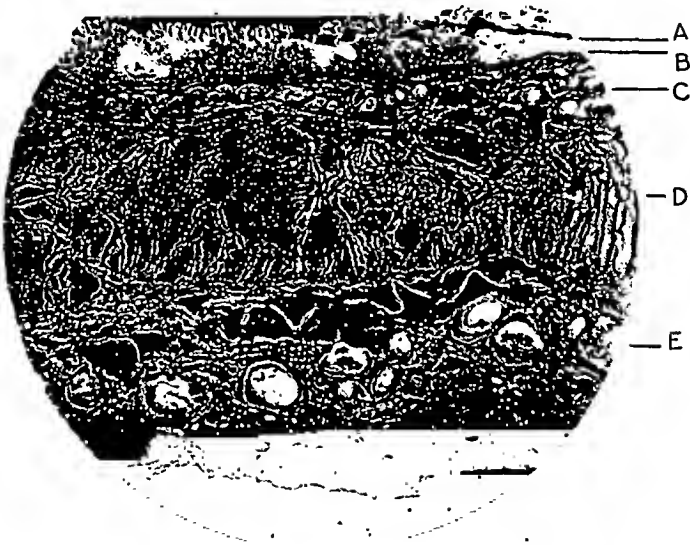


FIG. 21.—Chronic follicular gastritis, advanced stage. A, Mucous membrane densely infiltrated with inflammatory cells, and showing lymph follicles with germinal centres; B, Thickened muscularis mucosae; C, Fibrosis of submucous area and many dilated blood-vessels; D, Normal muscularis propria; E, Dilated blood-vessels in the subserous layer.

It is suggested by Faber that achylia gastrica may result from the extensive atrophy of the secreting elements in continued gastritis. While this may be true, it is quite certain the achlorhydria may occur in cases where very little atrophy can be found in the sections. In two of the cases here reported (*Cases 1 and 7*) hydrochloric acid was not detected in the stomach contents, but the glands showed little atrophy under the microscope.

*Thickening of the muscularis mucosae* is a constant finding in chronic follicular gastritis. Instead of being a thin compact sheet of muscle tissue this structure becomes thickened by hypertrophy, hyperplasia, and fibrosis. It may also be split into layers, between which are seen the same types of

inflammatory cells as are found in the mucosa. Beneath the muscularis mucosæ the inflammatory cellular exudate is much less pronounced. Islands of round cells, polymorphs, and eosinophils are commonly found at intervals in the submucosa, between the muscle layers of the muscularis propria, and even in the subserosa layer, but they are not always present. A small blood-vessel is generally to be found in the neighbourhood of each collection of cells. The small aggregations of cells in the subserosa probably precede the formation of the perigastric adhesions so frequently found at operation.



FIG. 22.—Chronic follicular gastritis, late stage, showing atrophy and cyst formation in gastric mucous membrane.

A fairly constant and striking feature of the lesion is the apparent increase in number and the distension of blood-vessels in the submucous and subserous layers. The small blood-channels of the mucous membrane itself are also frequently very prominent. These vascular features help to explain not only the hyperæmia of the peritoneal surface observed at operation, but also the occurrence of bleeding into the stomach. Areas of œdema in the muscularis propria are to be recognized in some of the specimens.

The small *hemorrhagic erosions* of the mucous membrane are of interest in connection with the gastrostaxis that may accompany gastritis. Histologically, the erosions consist of narrow clefts which cut through the surface at right angles, and extend for varying distances into the mucous membrane.

They occur either at the summit of small elevations and lead down into lymph follicles or their neighbourhood. or they are found in deep clefts between rounded elevations of the mucosa and extend downward as deep as the muscularis mucosæ. Their edges are always sharp and well defined. Superficial necrosis in the walls of these clefts does not appear to be a prominent feature. In this series they have not been found to communicate with the lumen of blood-vessels. The general appearance of the erosions is that of relatively small and unimportant defects, but massive bleeding and even



FIG. 23.—Chronic follicular gastritis, showing superficial erosion of the mucous membrane, leading down to a lymph follicle.

fatal hæmorrhage have been reported. Superficial ulcerations of the uppermost layers of the mucosa may occur in the acute ulcerative form of gastritis.

A relationship between hæmorrhagic erosion or superficial ulcer on the one hand and chronic peptic (gastric) ulcer on the other has not yet been established beyond question. Aschoff, in his theory of "mechanical-functional disposition of the gastric pathway", suggests the possibility of transition of erosions of the gastric street and isthmus ventriculi to true callous ulcer. Absolute proof of such an event is yet to be brought forward. That it is possible cannot be gainsaid, but that chronic ulcers invariably begin as erosions is quite another thesis, and must await proof.

Chronic duodenitis frequently accompanies chronic follicular gastritis.

There are some features in its gross and microscopic pathology which suggest a close relationship to gastritis, but for the purpose of the present study it is considered a separate though possibly an allied disease process.

### COMPLICATIONS.

From the standpoint of pathological anatomy there are six chief complications or sequelæ of chronic gastritis—viz., hæmorrhage, pyloric stenosis, gastritis polyposa, atrophic pangastritis, perigastritis with adhesions, and cancer.

In the absence of accurate knowledge on the point, *hæmorrhage* into the gastric lumen is attributed to erosion or superficial ulceration of the mucosa.

*Pyloric stenosis*, with retention, frequently found clinically, may depend upon hypertrophy and hyperplasia of the muscle, pylorospasm, polypoid overgrowth of the mucous membrane, or a combination of any two or all three of these factors.

In *gastritis polyposa* the visible projections of gastric mucous membrane are composed of hyperplastic epithelial masses, or small adenomas with an epithelial covering. This form of gastritis is one of the best known, and diagnosis by X-ray is not difficult. Hæmorrhage, pyloric obstruction, and cancerous growth are its serious complications.

*Atrophy of the mucosal cells* can be found in almost every case of chronic follicular gastritis. In the acute forms, and in the ulcerating and bleeding types, it is not a prominent feature, but in the advanced cases it can nearly always be demonstrated. The atrophy is patchy in distribution, and cysts are present. Faber teaches that the natural course of gastritis is first hypersecretion, later gradual atrophy, finally achylia. As mentioned above, it is not necessary for the whole mucous membrane to be atrophic before achylia sets in, but, on the other hand, when achylia develops it is certain that a very considerable part of the secreting tissue has been destroyed. In late cases the whole stomach shows atrophy (pangastritis). The disease is not then confined to the pylorus, as in follicular gastritis, but involves the whole organ. As atrophy proceeds achylia becomes more pronounced. Free acid disappears first from the stomach contents, then pepsin, and finally rennin. From a clinical point of view a cure is possible as long as rennin can be detected in the gastric juice.

In four of the nine cases here reported *perigastric adhesions* were found (perigastritis adhesiva). They were most prominent about the pylorus in all cases, but in one (*Case 8*) the duodenum also showed organized adhesions to the surrounding structures. The microscopic sections from *Case 3* are instructive in this connection. They show definite nests of inflammatory cells (polymorphs and round cells) between the strata of the muscular coat, and even in the subserosa. The inflammatory process may apparently penetrate the gastric wall, and lead to localized peritonitis resulting in adhesions.

### DIAGNOSIS, PROGNOSIS, AND TREATMENT.

**Diagnosis.**—The presence of chronic follicular gastritis is to be suspected in cases with a long clinical history of indigestion accompanied by hæmorrhage, if no ulcer or cancer can be demonstrated by the X rays. If there

is hyperacidity in the early stages, or anacidity later, the suspicion is strengthened. A slight retention makes the diagnosis fairly certain. Temporary improvement under conservative measures, with sudden recurrence of bleeding or bouts of pain, serve further to confirm the opinion. The diagnosis is made fairly certain if inspection of the stomach after the abdomen is opened reveals perigastric adhesions, reddening of the serous coat, hardening and thickening of the walls in the pyloric region, or enlarged soft lymph-glands. A positive diagnosis, however, can only be made with the aid of the microscope.

#### DIFFERENTIAL DIAGNOSIS.—

1. *Chronic gastric ulcer* is differentiated from chronic follicular gastritis by the presence of epigastric tenderness, and by craters in the X-ray films.

2. *Gastric cancer* will nearly always be detected by X rays. Konjetzny's two cases of incipient cancer in the mucosa of patients suffering from chronic follicular gastritis are strong evidence that cachexia of sudden onset in such cases should be interpreted as heralding the carcinomatous change, and treated accordingly.

3. *Œsophageal varix* may be ruled out by consideration of the clinical history, and by abdominal examination.

**Prognosis.**—It is quite possible that the disease may exist for decades. The usual expectation is that it will last for many years unless hæmorrhage, cancer, chronic ulcer, pyloric obstruction, or achylia should modify the course. The underlying pathology is chronic inflammation, and there is an undetermined psycho-nervous element superadded. Psychic effects (e.g., distaste for work, headache, loss of sleep, vague indigestion, or actual abdominal pain) play an important and often predominant part in the clinical picture.

#### Treatment.—

**CONSERVATIVE TREATMENT.**—When this is attempted it is carried out on a symptomatic basis—for example, bland diet, alkali for hyperacidity, hydrochloric acid for anacidity, gastric lavage for retention, bitter tonics for anorexia, belladonna for pyloric spasm, and purgatives if necessary for constipation.

**OPERATIVE TREATMENT.**—It goes without saying that, in *secondary gastritis*, the primary lesions should always be treated. The secondary gastritis may accompany chronic ulcer, cancer, neurasthenia, syphilis, hernia, gastro-ptosis, pylorospasm, tabes, pulmonary tuberculosis, neurosis, perigastritis, previous gastro-enterostomy or partial gastrectomy.

Without entering into an argument of conservative treatment *v.* operative treatment, suffice it to say that our present knowledge is built up from operative cases, the data of which are complete and available. The case in favour of conservative treatment is yet to be made. Wanke treated by conservative measures 22 cases in which a normal-appearing stomach had been found at operation in cases of gastritis: 4 were cured (19 per cent), 8 were improved (36 per cent), and 10 failed (45 per cent).

In *primary gastritis* gastro-enterostomy has few supporters. In two cases in the present series this operation failed, and subsequently pylorotomy had to be undertaken to effect a cure.

*Extramucosal pyloromyotomy*, with which the name of Payr is associated, was introduced by him for treatment of those cases without ulcer which

showed true pylorospasm, with or without gastrostaxis or delayed emptying. When used in 16 selected cases (Payr, 1925), 12 were very well or fairly well, 2 improved, 2 not improved; 1 case died of phlegmonous gastritis.

*Resection* of the pyloric portion of the stomach and first part of the duodenum is supported by Konjetzny, Finsterer, Flörekén, Elter, and Nicolaysen (*Table II*). The continuity of the tract is re-established by

*Table II.*—RESULTS OF PARTIAL GASTRECTOMY IN CHRONIC GASTRITIS.

NAME	DATE	INTERVAL SINCE OPERATION	TOTAL	CURED	IMPROVED	FAILED
Elter .. ..	1925	Over 1 yr.	15	12	2	1
Wanke .. ..	1929	Up to 4 yrs.	13	5	2	6
Nicolaysen .. ..	1928	1 yr.	7	7	—	—
Finsterer .. ..	1924	—	35	35	—	—
Paaby .. ..	1927	—	3	3	—	—
Konjetzny .. ..	1924	6 wks. to 10 mos.	8	8	—	—
This series .. ..	1930	6 wks. to 29 mos.	9	7	2	—
Totals ..			90	77	6	7

Billroth I, or by the Polyá, Moynihan, Balfour, or Hoffmeister modifications of the Billroth II procedure. Wanke gives a warning that cases resected early in the disease are more likely to have post-operative trouble (fundus gastritis) than those operated upon late, when the inflammation is confined to the pyloric region. In the 9 cases of this series the operation known as the Polyá modification of the Billroth II procedure was carried out in 6 cases; in 2 the pyloric portion of the stomach and the first part of the duodenum were removed, (pylorectomy), leaving intact a previously existing gastro-enterostomy; in 1 case the Balfour method of partial gastrectomy was employed (*Table III*).

*Table III.*—RESULTS OF RESECTIONS AND OF PYLORECTOMY IN CHRONIC GASTRITIS IN THE PRESENT SERIES.

OPERATION	TOTAL	CURED	IMPROVED
Partial gastrectomy	7	5	2
Pylorectomy ..	2	2	—
Totals ..	9	7	2

### SUMMARY.

In the nine cases of chronic follicular gastritis reported above there were symptoms or signs suggesting chronic ulcer, but no ulcer was found at operation or in the gross specimen. In all cases histological study of the portion of the stomach removed at operation showed a characteristic lesion, of which the most prominent finding was infiltration of the mucous membrane by inflammatory cells, in places forming large *follicles with germinal centres*.

Critical study of the results of partial gastrectomy in the present series and in the cases in the literature shows that the operation cures or gives relief in most cases, and fails only in a small number. Until the etiology is known partial gastrectomy (Billroth I or modified Billroth II) should be the treatment of choice when conservative measures fail. Pyloromyotomy probably has a place in cases where pylorospasm is present, although it will not guarantee freedom from the possibility of subsequent hæmorrhage or cancer.

This study was carried out in the Department of Pathology of the Montreal General Hospital, under the direction of Dr. L. J. Rhea, and the cases are reported by the kind permission of Dr. A. T. Bazin, Dr. E. M. Eberts, Dr. W. L. Barlow, Dr. C. K. P. Henry, and Dr. A. Stewart, of the Surgical Staff.

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**CHONDROSARCOMA OF THE FIBULA.**

BY A. VICTOR NEALE AND E. D. ALLEN-PRICE, BIRMINGHAM.

EWING<sup>1</sup> states that the cellular chondromas are comparatively benign neoplasms, and conservative local treatment often proves satisfactory. Osteoid chondroma is a peculiar form of skeletal chondroma arising beneath the periosteum of long bones. As the growth enlarges, the bone assumes a thickened spindle outline simulating in form some of the osteosarcomas. A tumour which grows very slowly is probably of low malignancy, but may change its character and grow rapidly. Slowly growing local tumours may produce metastases early. Encapsulation is an important factor in the prognosis of the sarcomas occurring in relation to bones. Phemister<sup>2</sup> has recently recorded some observations concerning chondrosarcoma, and he is inclined to place this type of neoplasm in a separate group. Cartilage may be seen in ossifying sarcoma of bone, while the tumour may contain cartilage alone and show no metamorphosis.

Simple chondromas which originate on the surface of a bone or beneath the cortex and subsequently break through to the surface are likely to possess islands and branching areas of calcification. A case of this nature is recorded by Phemister :—

Male, age 14. Large swelling in the upper portion of the right leg which had developed for nine months with increase of pain. Physical examination was otherwise negative and the chest clear. X rays showed a large oval swelling in the upper seven inches of the right tibia. At operation the femoral vein was found to contain a column of cartilage, and microscopically the tumour consisted of hyaline cartilage with extensive areas of necrotic degeneration. Nine months later the boy was in excellent health.

True chondrosarcoma consists of islands of hyaline cartilage, areas rich in mitotic figures, and older calcified or ossified portions, the calcification being irregular and blotchy, giving a characteristic radiographic picture. Usually metastases occur later than with other types of sarcoma; there is a tendency to invade veins, and metastases are cartilaginous. Phemister records that out of 61 cases of bone sarcoma, 10 were chondrosarcoma, the tumour having occurred in the femur, humerus, tibia, maxilla, spine, or rib. No recorded instance has been found of this type of tumour occurring in the fibula.

Chondrosarcoma may arise centrally or peripherally. The central type causes reactionary bone to form around, whereas the peripheral type does not cause bone reaction but gives a typical X-ray picture of a tumour with scattered areas of calcification.

Details of an interesting case of chondrosarcoma are given by Phemister :—

Male, age 40. The patient had noticed an outgrowth on the lateral aspect of the right tibia for about thirteen years. At the age of 27 this mass began to increase



in size, and at 34 it was as large as a fist. The neoplasm was excised locally, but it recurred, and at the age of 37 further operations were performed. Microscopically this tumour at the first operation consisted of apparently innocent cartilage. The tumour recurred again twice locally, and amputation was eventually necessary. Finally the tumour was found to be richly cellular, with karyokinetic figures very suggestive of malignancy.

This case resembles the one observed by us in the apparent innocency of the primary tumour and recurrence in malignant form after removal.

**HISTORY.**—A boy, age 16 years, stated that three years previously he was bitten by a gnat on the outer side of the right leg. A swelling developed here, and exercise



FIG. 24.—Radiogram taken in 1927, showing the calcific changes in the neoplasm.



FIG. 25.—Radiogram taken in 1930, showing the more diffuse nature of the tumour.

caused considerable pain. The swelling and pain increased and brought him to hospital.

**ON EXAMINATION.**—He was a healthy-looking boy and nothing abnormal was found except a lump on the outer side of the right leg. The lump was very hard and fixed to the fibula. It was  $3\frac{1}{2}$  in. long and 2 in. broad, with the centre at the junction of the middle and lower thirds of the fibula. Pressure on the lump, and flexion and extension of the ankle-joint, caused pain. He stated that recently the pain had been continuous and worse at night.

X rays (*Fig. 24*) showed that the tumour was on the surface of the fibula and

contained calcified areas. Its general appearance suggested malignancy. Radiographs of the chest showed no signs of metastasis.

**FIRST OPERATION.**—Local removal of the tumour was carried out in November, 1927. When exposed it appeared to be encapsuled. The tumour, together with the shaft of the fibula, was excised, the division of the latter being made 2 in. above and below the extremities of the growth.

**SUBSEQUENT PROGRESS.**—For two and a half years the patient remained well, led a normal active life, and grew 6 in. in height. In April, 1930, he again noticed a swelling at the site of the operation. This increased in size, and in August, 1930, after a trivial injury, continuous pain recommenced and he returned to hospital. A tender swelling in the original situation was present, and X rays revealed active sarcoma occupying the gap between the fragments of the fibula (*Fig. 25*). A Wassermann test was negative, and no metastases could be found.

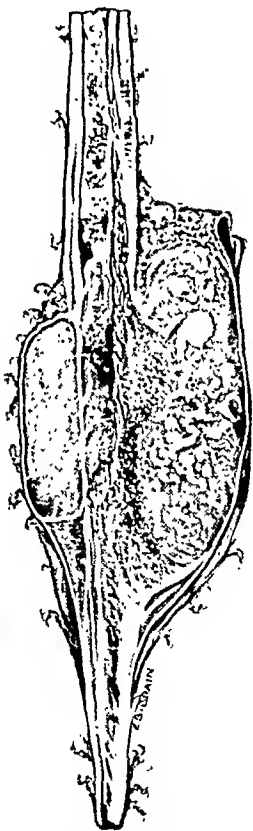


FIG. 26.—Tumour removed at first operation (November, 1927).



FIG. 27.—Recurrent tumour as seen at second operation (September, 1930).

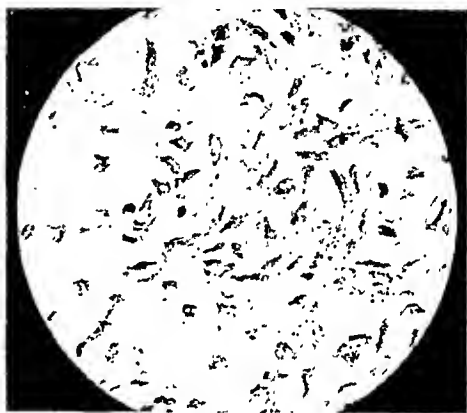
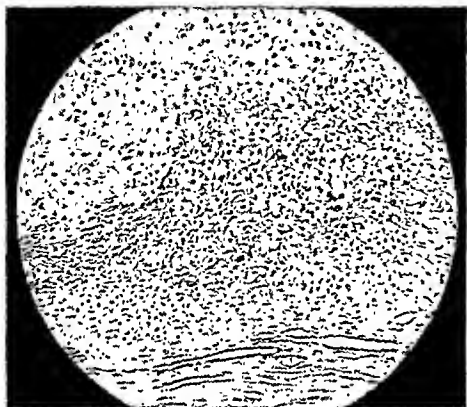
**SECOND OPERATION.**—Amputation through the lower part of the thigh was performed. Three months later the boy was in excellent health with no signs of metastasis.

**PATHOLOGICAL REPORTS** (*Figs. 26, 27*).—

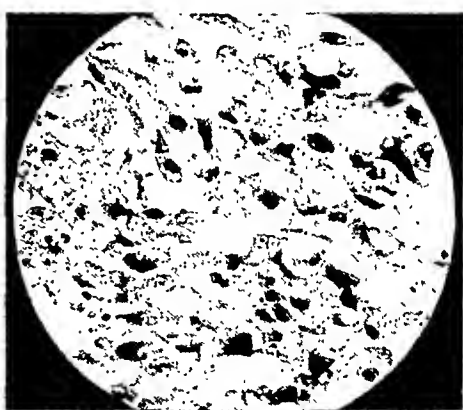
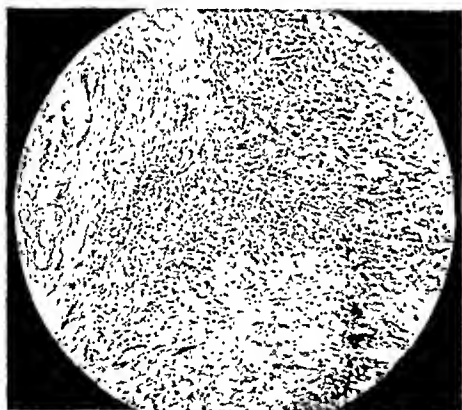
1. The tumour removed in 1927 was cylindrical in shape and had an indefinite fibrous capsule. It was firmly attached to the fibula and cartilaginous in structure.

Here and there was evidence of commencing penetration of the cortex of the bone, and, microscopically, in places there were areas of cell activity suggestive of early malignancy.

2. The recurrent tumour was an obvious chondrosarcoma, with definite cell activity and invasive character. The cells showed many mitotic figures, increased chromatism, and great variability in outline. Careful dissection did not reveal any infiltration of the soft tissues, and it still appeared to be confined to the bony structures.



FIGS. 28, 29.—Low- and high-power views of tumour removed in November, 1927.



FIGS. 30, 31.—Low- and high-power views of recurrent tumour removed in September, 1930.

An interesting feature of the case was the discovery of very early malignant changes in the first tumour and their more marked character in the recurring one. The essential origin of the tumour in cartilage is shown in the microphotographs (Figs. 28-31).

This case, like Phemister's, illustrates that sarcomatous change in a chondroma may be very gradual and probably remain within the bounds of cure by amputation for a moderate period of time (apparently about three years in the cases observed).

Chondrosarcoma may, it appears, remain amongst the less malignant sarcomatous tumours of bones, but no doubt too great a latitude cannot be allowed, because rapidly increasing infiltrative and metastatic capacities may arise at any time. It would probably have been a safer procedure, in the case outlined above, to have carried out amputation in the first instance. The very unusual situation of the neoplasm—namely, in the fibula, a site apparently hitherto unobserved—is of additional interest.

This case was under the care of Mr. Bertram Lloyd in the wards of the Queen's Hospital, Birmingham, and we are grateful to him for permission to record these observations. The microphotographs were kindly supplied by Dr. J. F. D. Shrewsbury.

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## THE TIME FACTOR DURING WHICH VASCULAR RESPONSES PERSIST FOLLOWING PERI-ARTERIAL SYMPATHECTOMY.\*

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A CURIOUS feature of the vascular responses to extirpation of the sympathetic peri-arterial network is their transient nature. In experiments designed to ascertain what vascular changes, if any, follow this operation, Hemingway and I have shown that in the cat and the rabbit peri-arterial sympathetic destruction increases local circulation, but that its effects are exceedingly transient.<sup>1</sup> A readjustment, the mechanism of which is at present obscure, comes into action at a time which is variable according to species and possibly also the vessel selected and the site of the particular vessel operated upon—for example, we found the readjustment in the case of the femoral artery of the cat to take place in a matter of seconds, whereas that following extirpation of the nerve net on the carotid of the rabbit persisted for some hours or days. This readjustment appears to follow also extirpation operations on the main sympathetic system. Thus we have noticed that vasodilatation in the ear of the albino rabbit consequent upon cervical sympathetic neurectomy is slowly recovered from, but have also observed that its effects are both greater and more lasting than the peri-arterial operation performed on the carotid artery.

A recent case throws some light on the probable duration of the time factor in man :—

**HISTORY.**—The patient was a steel worker, age 61, who had been an international footballer and was a healthy man with a comparatively healthy vascular system and a negative blood Wassermann reaction. Just over five years before admission his left leg had been severely scalded with hot paraffin. The leg ulcerated and had never healed since. For one year he attended three times a week for dressings at a hospital out-patient department. He had had long periods of rest, but with very little effect.

**ON EXAMINATION.**—Two ulcers were seen, one on the anterior surface of the leg in the middle third, the other, slightly larger, on the calf. The leg was discoloured brown, and the great toe showed onychogryposis.

**OPERATION** (Dec. 4, 1930).—Under chloroform and ether anaesthesia, the left femoral artery was exposed in Hunter's canal and found to be a healthy vessel. It was accompanied by two large nerves regarded as the internal saphenous and the nerve to the vastus internus, each as large as a normal ulnar nerve. These nerves were not lobulated, but uniformly enlarged, and appeared to be merely hypertrophied. The phenomenon was unique in the experience of those present in the theatre. The artery was stripped of its adventitia over a length of half an inch, and over this area and for a further quarter of an inch above and below was well

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\* From the Surgical Unit, the Welsh National School of Medicine, Royal Infirmary, Cardiff.

painted with strong carbolic acid, a method which we had previously applied effectively and without detriment to the artery in animals. The deep fascia was closed with catgut and the skin with clips.

**AFTER-HISTORY.**—On the morning following the operation the left foot—that is the one on the side of the denervated femoral artery—seemed distinctly warmer to the touch than the right, and the patient complained of his right toes being cold while those of the left foot were warm. The epidermis of the whole of the leg next began to break up into scales, which gradually fell off, and the ulcers commenced to heal. By the thirty-ninth day the ulcer on the front of the leg had soundly healed and the whole leg had peeled, leaving healthy skin beneath. The ulcer on the calf had almost healed, but now seemed to be stationary. By the forty-fourth day the calf ulcer had not only ceased to heal but was definitely getting larger.\* The anterior ulcer remained healed.

It thus appeared as if a falling off in the beneficial effects which were observed almost immediately to follow the operation took place somewhere about five weeks subsequent to the operation.

### COMMENTARY.

The effects of the peri-arterial sympathectomy—namely, increased warmth, the scaling, and the reparative changes in the ulcers—were regarded as expressions of an increase in local circulation, the scaling being looked upon as due to proliferation and renewal of the stratum corneum consequent upon increased activity of the stratum cylindricum of the epidermis. As estimated by the condition of the ulcers, the response persisted for approximately five weeks, after which time its effects were no longer apparent. It would appear, therefore, that in man, as in animals, an increase in local circulation follows peri-arterial sympathectomy applied to the femoral artery, but that the increase is likewise a transient condition, lasting, however, for approximately five weeks. This period contrasts with those of a few seconds to as many days observed by Hemingway and the writer in plethysmographic and other experiments carried out upon cats and rabbits, and the wide variation in the length of the period in different species recalls the wide range of the duration of Monakow's diaschisis in animals and in man. It is regretted that a series of cases such as that from which these observations have been made is not to hand to enable a more precise and average measurement of the time interval to be made, but it is not often that cases as suitable as that here detailed present themselves for study subsequent to the operation, the majority either being discharged from hospital or coming to amputation of the limb before the post-operative reaction phase has terminated.

From observations made by the use of Pachon's oscillo-sphygmomanometer on cases in his clinic, Professor René Leriche<sup>2</sup> gave the writer to understand that he believes that the vasodilatation lasts for somewhere about the same time as that observed on purely clinical grounds in the case here recorded, but Dr. Leriche commented upon the difficulty of satisfactorily measuring vascular responses except by plethysmography. It may be concluded by analogy with the experimental work previously referred to that a

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\* By skin-grafting, this ulcer subsequently healed and the patient was discharged. He was seen a fortnight after his discharge, and had a soundly healed leg, but this is irrelevant as far as the thesis of this paper is concerned.

greater and more lasting increase in local circulation follows the extirpation of the sympathetic supply to the limb (by ramisection or ganglionectomy), but that from the observations here recorded, a vasodilatation lasting somewhere about five weeks will follow a successful peri-arterial sympathectomy applied to a healthy artery supplying a healthy capillary bed, and that in certain cases in which a transient vasodilatation of this duration will be beneficial, the operation may with advantage be considered.

The writer is much indebted to the Medical Research Council for partly defraying the expenses in connection with the experimental work referred to in this note.

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## PAPILLIFEROUS CYSTOMA OF THE PETROUS BONE ASSOCIATED WITH HYPERNEPHROMA AND CYSTIC PANCREAS.\*

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IN a monograph on cerebellar cysts, Lindau<sup>1</sup> (1926) drew attention to the not infrequent coexistence of hæmangioblastomas of the central nervous system, especially of the cerebellum, with retinal angioma (von Hippel's disease); but in addition to this there have appeared fairly commonly associated lesions in other parts of the body. Thus in his series of 40 cases Lindau reported cystic pancreas in 8, renal cysts in 10, and hypernephroma in 6; cystic epididymis has also been noted. In the case we have to report the main lesions were a papilliferous cystoma of the right petrous bone, hypernephroma of the right kidney, cystic pancreas, and cysts in the right epididymis. While the tumour of the petrous bone was intrinsically interesting—so far as we can discover no exactly similar growth in this situation has been recorded—the associated lesions call for some comment, especially because there is a tendency to regard cystic pancreas as a pathognomonic feature of Lindau's disease.

### CASE REPORT.

**CLINICAL HISTORY.**—J. B., male, age 23 years, was admitted to the Neurological Surgical Service of the Manchester Royal Infirmary at the instance of our colleague, Professor Tylecote, with the diagnosis of intracranial tumour, probably acoustic neuroma. Some twelve months before admission the patient had developed a slight weakness of the right facial musculature, which was the only symptom until, some months later, hearing became less acute in the right ear. Both of these disabilities steadily but slowly progressed, and on admission (July 5, 1929) a complete right facial paralysis was manifest, and deafness in the right ear, almost but not quite complete. During the previous two months headache was intense, and latterly had been accompanied by vomiting of such a degree as to cause great emaciation. The patient had become dizzy, and reeled to the right when he walked; on one or two occasions he had fallen to the right.

**ON EXAMINATION.**—The patient presented the following signs: (1) Right facial paralysis, complete peripheral type; (2) Deafness in right ear with an unperforated tympanum; (3) Slight choke of the right optic nerve-head, venous congestion only on the left, no retinal angioma seen; (4) Nystagmus on fixation to the right; (5) No clear evidence in the limb musculature of cerebellar involvement. The subsequent autopsy showed that the cerebellum was neither infiltrated

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\* From the Department of Pathology, the Victoria University of Manchester, and the Manchester Royal Infirmary.



nor materially indented by the growth. The patient held his head flexed forward, and rotated slightly to the left. The diagnosis was not absolutely clear. The fact that the facial paralysis led in the symptomatology seemed to make it unlikely, but not impossible, that the tumour was an acoustic neuroma. We have had a series of these tumours in which deafness was not absolute, the tumour presumably originating in the sheath of the vestibular division, as Henschen contended. A basal endothelioma appeared more probable.

The question of treatment became one of some urgency because vomiting was now very distressing, and during the last few days there had been some difficulty in swallowing. At the same time the facial paralysis, the fixed posture of the head, the violence of the headache and vomiting, all combined to make a picture of unforgettable misery. The patient's skin was dry, and it seemed very unlikely that he would survive a lengthy or bloody operation.

OPERATION.—It was decided therefore to limit any immediate operative procedure to a decompression, with observation of the local condition, leaving the

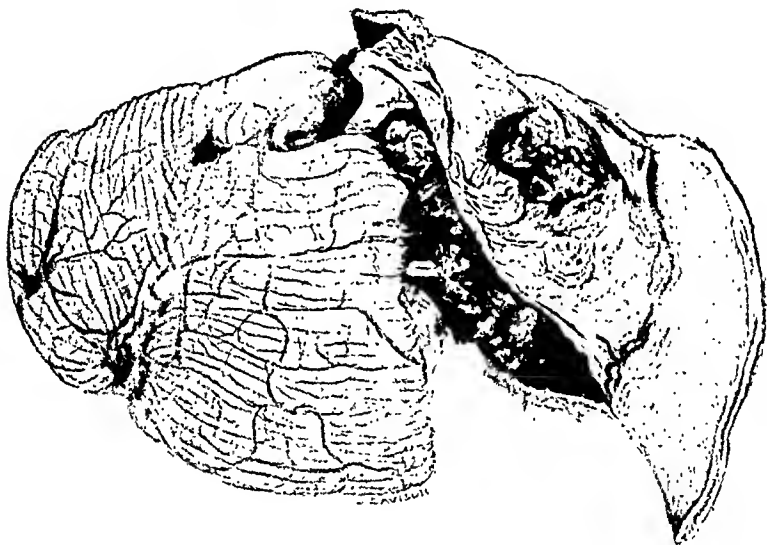


FIG. 32.—Tumour of right petrous bone seen from above, cerebellum retracted.

removal of the tumour for another session, unless it should promise to be unexpectedly easy. Accordingly on June 6 a wide suboccipital exploration was performed by the usual technique, very considerable foraminal herniation rendering laminectomy of the arch of the atlas imperative. The right cerebellar hemisphere was gently retracted and the vagal and facial exits were inspected. Dark-red, hard tumour with no clear boundary was encountered on the back of the petrous bone, and nothing further was done. The wound was closed in the usual careful fashion. The patient's condition was unaltered by this intervention; he did not cease to vomit, and despite the intravenous administration of glucose and hypertonic saline, he died of pneumonia five days after the operation.

AUTOPSY FINDINGS.—The anatomical diagnosis was: Tumour of the right petrous bone indenting cerebellum but not invading it (*Fig. 32*); cysts in the pancreas; encapsulated tumour of right kidney; cysts in epididymis; bilateral bronchopneumonia. The pancreas contained a few cysts 0.5 to 2 cm. in diameter. The right kidney appeared normal in general aspect, but on the middle of its outer border there was a round encapsulated growth, 9 mm. in diameter, projecting under the renal capsule, which on section showed yellow patches alternating with red. In

## PAPILLIFEROUS CYSTOMA OF PETROUS BONE 57

the right epididymis there were a few hard cysts 0·75 cm. in diameter around the globus major, and a similar hard mass in the spermatic cord above. The vertex of the skull was normal. The dura was normal as seen from above. The pia and arachnoid were moderately congested. On removing the brain a nodulated dark-coloured tumour was seen beneath the dura covering the right petrous bone, the membrane being much raised and invaded. The right temporosphenoidal lobe was lightly adherent at one point about the tegmen tympani. The main body of the growth was seen to occupy the centre of the petrous bone, a fairly large extension passing upwards through the region of the tegmen. Similar extensions passed backwards and inwards from the region of the internal auditory meatus. The latter extension came into close relation with the cerebellum and pons in the region of the auditory nerve, but the relation to the nerve itself was not clear. On section through a portion of the tumour, its substance was found to be very dark in colour, and there was an appearance of numerous minute cavities filled by dark material. This suggested to the naked eye that the growth might possibly be an angioma.

There was no gross abnormality of the pericardium, heart, œsophagus, thyroid, stomach, liver, spleen, testes, or left kidney.

**DISSECTION OF THE RIGHT TEMPORAL REGION.**—On drilling through the cortex covering the mastoid air-cells, a large isolated air-cell 1·5 by 1 cm. was soon entered. It was entirely uninvaded by tumour. The rest of the mastoid air-cells, and the mastoid antrum, were filled by gelatinous-looking tumour material. The bone forming the anterior wall of the external auditory meatus was nibbled away with forceps until the tympanic membrane was displayed. This was dark in colour and not obviously perforated. Certainly no tumour extruded through it. On removing further bone, the specimen could be opened in a horizontal direction into an upper and lower half, and the tympanic cavity was seen to be entirely filled by tumour. The growth could be easily separated from the tympanic membrane, in which two or three minute openings were seen after its removal. There seemed to be little doubt, however, that the tumour was not intimately connected with the membrane, because of the ease with which they were separated. We received the impression that the tumour had not arisen from the walls of the middle ear and that such erosion as was present was of a secondary nature. If it had originated here, the structures of the cavity would have been more profoundly altered. The tumour occupying the middle ear, the tympanic membrane, the malleus, and a specimen consisting of a small group of mastoid air-cells, were preserved for histological examination. The internal ear was then removed as far as possible *en bloc* for decalcification. The cut passed through the external semicircular canal, whence fluid exuded. No gross tumour material was seen, the internal ear giving the impression before it was opened of being free from growth. In general, the whole petrous bone, as far as its tip, seemed to be infiltrated. The carotid canal was not involved, and the artery was normal.

**HISTOLOGICAL EXAMINATION.**—On histological examination tumour tissue was found in the mastoid air-cells, the internal ear, the tympanic cavity, and the malleus. The tympanic membrane was uninvaded by tumour. The tumour presented the same general appearance in these various sites, and may be described as a papilliferous cystoma.

The cysts were closely packed and irregular in shape, varying from 0·8 to 5 mm. in diameter. They were bounded by a single layer of flat to low columnar cells. The fibrocellular stroma supporting this layer of cells varied much in amount, in some areas being very scanty, in others abundant and dura-like (*Fig. 33*). In the dense parts of the stroma there were a few globular laminated concretions staining darkly with hæmalum, and occasional deposits of cholesterol. The cysts were filled with homogeneous hyaline material staining deeply with eosin, in which mucin could not be demonstrated by staining with mucicarmine, thionin, or methylene blue. Areas of hæmorrhage were visible towards the walls of many of the cysts, being on the whole most marked in cysts in which papillary ingrowths were few or absent. Groups of round cells, about 25  $\mu$  in diameter, laden with brown pigment, were seen in these hæmorrhagic areas.

The papillary ingrowths lay embedded in the hyaline contents of the cysts (*Fig. 34*). In the specimen from the middle ear the papillæ were so abundant that



FIG. 33.—Tumour of right petrous bone, showing dense fibrous stroma surrounding the cysts, in one of which papillæ are visible. ( $\times 50$ .)



FIG. 34.—Tumour of right petrous bone, showing branching papillæ embedded in hyaline material. ( $\times 100$ .)

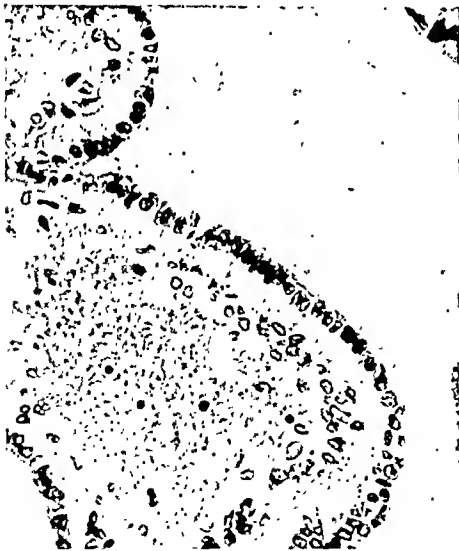


FIG. 35.—Tumour of right petrous bone, showing part of a papilla whose covering cells have nuclei at their free poles. The vascular stroma is clearly seen. ( $\times 200$ .)

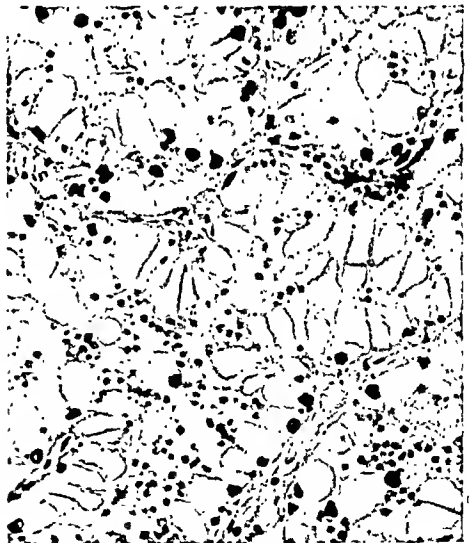


FIG. 36.—Tumour of right kidney, showing the sharp definition of the cells and their small round nuclei. ( $\times 200$ .)

the cystic structure of the tumour was almost masked, the hyaline material being very scanty, though showing the features described where it could be found. The

papillæ were composed of a core of branching fibrocellular stroma continuous with that surrounding the cysts, but relatively more vascular, covered by a single layer of low—rarely tall—columnar cells. Direct continuity between the cells covering the papillæ and those lining the cysts was established in places. The cytoplasm of the cells covering the papillæ was clear, and their nuclei large, oval, or cuboidal in shape, and containing a moderate amount of chromatin. The nuclei often lay at the free border of the cells, giving the growth a striking appearance (*Fig. 35*). This feature was best seen where the cells were tallest. In some of the cells appropriate examination showed the presence of fat and doubly-refracting bodies, which occurred irregularly in scattered patches. The specimen of the internal ear and malleus showed invasion and destruction of the bone, the tumour itself retaining its characteristic morphology. The peripheral nuclei of tall columnar cells forming the covering of the papillæ were particularly well seen in the internal ear. It was possible to recognize the organ of Corti in places.

The tumour of the right kidney was encapsulated by fibrous connective tissue. Within this were enclosed acini and solid alveoli of epithelial cells supported by a scanty delicate vascular stroma (*Fig. 36*). The cells were fairly large, of cuboidal or low columnar shape, with sharply defined walls, small round nuclei, and clear bodies. By suitable staining they were found to contain abundant neutral fat, some doubly-refracting fat, and abundant glycogen. The growth corresponded in structure with the Grawitz tumour of the kidney or hypernephroma, but there was no evidence to show that it had penetrated its capsule or that it was in active growth.

The cysts in the pancreas were lined by a single layer of cuboidal cells with spherical nuclei. Careful examination failed to demonstrate any neoplastic process in these cysts.

### DISCUSSION.

Objection may be taken to our use of the term 'cystoma', admittedly a purely morphological designation. The cells composing this tumour were clearly epithelial, but of such peculiar type that it is impossible to be certain exactly whence they had been derived. The tumour was composed of fully differentiated elements, and was structurally uniform. The epithelial cells, while varying much in height from place to place, were individually well defined units. Indications that the tumour was not recently formed were the presence of concretions, old hæmorrhages, sporadic deposits of cholesterol, and the fact that the fibrous stroma showed a marked preponderance of fibres over cells.

We are satisfied that the histological appearances definitely exclude an etiological relationship between the tumour of the petrous bone and that of the kidney. Each had a fairly uniform and distinctive structure, and no transitional stages between them could be found. The cystic structure, the hyaline material, the papillæ embedded in it, with their remarkable cubical or columnar epithelium with peripherally situated nuclei, and the dense stroma with concretions, were features peculiar to the petrous tumour. The renal tumour, on the other hand, presented a picture conforming in every way with the Grawitz tumour. Its acinar structure with solid alveoli of sharply defined cells containing small dot-like nuclei irregularly arranged, its uniformly delicate vascular stroma, its abundant glycogen, and its well-formed fibrous capsule, were without counterpart in the petrous tumour. Whereas there was no evidence of active growth in the renal tumour, the petrous tumour was seen to have invaded and destroyed bone.

In considering the possible intra-petrous sources of such a tumour as this, we have been led to review those recorded cases of primary carcinoma of the middle and internal ear whose structure in any way approached that of our own. Such cases are few, the most notable being those of Lange,<sup>2</sup> Furstenberg,<sup>3</sup> and Maestranzi.<sup>4</sup>

Lange<sup>2</sup> (1904) was the first to record a case of cylinder-celled carcinoma primary in the middle ear. The external auditory meatus was occupied by a polypoid growth that was firmly attached to the tympanic membrane but nowhere adherent to the meatal wall. The tumour caused widespread destruction of the petrous bone, and metastases were found in the regional lymph-glands, in those of the neck, and in both lungs. It was characterized by an abundant production of mucus as shown by metachromatic staining with a dilute thionin solution. Histologically it showed a simple epithelium composed mainly of columnar, partly of cubical, cells with basal nuclei, that formed the lining of spaces with mucous contents in which clumps of rounded cells lay free. The stroma was scanty. Calcium carbonate concretions were present in both primary and secondary growths. Accepted by Furstenberg<sup>3</sup> (1924) and Marx<sup>5</sup> (1926), the diagnosis was impugned by Schlittler<sup>6</sup> (1919), who pointed out that Lange failed to describe the histological structure of a submaxillary swelling noted at the autopsy, and that it is in consequence uncertain whether this swelling represented enlarged lymph-glands or a tumour of the submaxillary salivary gland. Schlittler maintained that the histological account of Lange's tumour was not incompatible with that of a deposit secondary to a tumour of the submaxillary salivary gland. That such tumours may be morphologically indistinguishable from carcinomas, and that they frequently exhibit degenerative changes of various kinds, has, according to Schlittler, been shown by Küttner.<sup>7</sup>

Furstenberg<sup>3</sup> described a primary adenocarcinoma of the middle ear and mastoid. The external auditory meatus was not affected, squamous epithelium being continuous over the tympanic membrane, which the growth had involved. The tumour was not uniform in structure, being alveolar in some parts and glandular in others. The glandular parts were very irregular. The abundant stroma gave the centre of the growth a scirrhus appearance. Warthin, who made the pathological examination, regarded the growth as primary in the middle ear, "representing a teratoid disturbance of development with blastomatous transformation".

Maestranzi<sup>4</sup> (1927) described a primary alveolar cancer of the middle ear. It presented as a polyp unattached to the wall of the external auditory meatus. Invested by stratified squamous epithelium, its substance contained alveolar cavities occupied by clumps of cells corresponding in type to cylindrical epithelium. Some cells lined the walls of the cavities, others lay free within them. In general appearance this tumour was not unlike that described by Furstenberg.

Of these tumours, the only one to which our petrous tumour bears any close resemblance is that of Lange. From this it differs both cytologically and histochemically, and also by the fact that it was the only growth of its kind in the body. From Lange's tumour, again, and also from Maestranzi's, it differs in that it did not penetrate the tympanic membrane, a point

against the view that our tumour might have arisen in the tympanic cavity. If the growth began in the internal ear, complete destruction of the latter might have been expected, in view of the extent of the growth in other parts.

The peripherally placed nuclei of the taller epithelial cells of the papillæ suggest a thyroid or parathyroid origin. No tumour was found in these organs at the autopsy, but that the petrous tumour could have started from an embryonal rest is not entirely impossible in view of the close developmental relationship subsisting between thyroid, parathyroids, and middle ear. The possibility of Lange's tumour having originated either from embryonal rests or from a primary growth in the thyroid does not appear to have been considered by Lange or by Schlittler.

We have finally to consider whether our petrous tumour might have been derived from neural epiblast, a suggestion made attractive by the discovery in the patient of a series of congenital lesions identical with those described by Lindau as occurring with hæmangioblastomas of the central nervous system. Our petrous tumour bore no resemblance to a hæmangioblastoma, and Lindau, to whom we submitted the slides, was of the same opinion. He suggested that our tumour was possibly collateral to the hæmangioblastomas of the central nervous system, and that it might have arisen from the anlage of the choroidal system, derived from the neural crest; the companion lesions would fortify this suggestion.

Hence it is possible that the lesions in our case may be the result of an error in development cognate with that which must underlie the lesions correlated by Lindau. In cases exhibiting multiple congenital lesions any one of them might elaborate itself into the clinically dominant feature. Pathological interest, however, centres in the developmental error rather than in any individual lesion that may declare it. It seems not unlikely that Lindau's disease, and the case we have described, are simply special forms of a more general disorder.

Whether cysts of the pancreas can be regarded as presumptive evidence of one particular type of tumour is in our opinion debatable. A highly cystic pancreas has been figured independently by Hadfield<sup>8</sup> (1929) in a recent report on a cerebellar angioma. Lindau<sup>9</sup> (1931) states that he does not think that these associated somatic anomalies have an angiomatous origin. It remains to be seen whether future observations will confirm the particularity of this association. Our own case throws doubt on it, though it must be admitted that the pancreas itself was not exactly of the type that Lindau regards as pathognomonic of his syndrome. Had our patient survived long enough, there is the possibility that the pancreas would eventually have conformed to type.

From the clinical standpoint it is clear enough how the symptoms in the case we have described came to mimic a posterior fossa tumour, and a more accurate diagnosis might have been made had the skiagrams, which actually showed changes in the texture of the petrous bone, been better interpreted. The posture of the patient, so important clinically, served to deviate the head out of the true antero-posterior axis in the skiagrams, so that a failure of upper contour was missed. The pathological study of

the growth, however, showed that its true nature could not have been determined clinically. Only once have we seen a similar erosion, and that was due to a cavernous angioma.

In conclusion it is our pleasant duty to thank Professor Lange, of Leipzig, and Dr. Lindau, of Lund, for personal communications, and Professor J. Shaw Dunn for his valued interest and counsel during the course of this study.

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## A CLINICAL STUDY OF ACUTE APPENDICITIS IN OLD AGE.

By J. LEWIN,

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It might well be supposed that the last word had been said on the subject of appendicitis, but during the past few years my attention has been drawn forcibly to a considerable number of cases of a uniform but very anomalous type occurring in people in advanced years. The picture presented is so constant that I find that such cases may be confidently diagnosed from the other conditions which they mimic. As they are not even mentioned in the text-books, I have ventured to present this short clinical study with the hope that it may be of some assistance to those who are confronted with similar cases. The anatomy and etiology of appendicitis will not be discussed, but consideration given only to the clinical manifestations of the disease which are peculiar to old age.

I am aware that any definition of old age must be, to a large extent, an arbitrary one. It is frequently stated that acute appendicitis is rare in old age, but in my own experience this is far from being the case, and though 60, which I make the lower limit, may seem somewhat drastic, it does include every case in my series, each of which is a manifestation of the declining power of the body to contend with bacterial infection. In the last three years I have had twelve such cases, in addition to at least as many more which presented symptoms comparable to those encountered in the large run of cases in young people. It is clear, therefore, that appendicitis in old people is far from being a rarity. Of my twelve cases, nine were men, and three were women, and their ages were: 62, 63, 64 (2 cases), 69 (2 cases), 71, 72, 73, 74, 75, and 86.

The underlying factor in all these cases is the alteration in the reaction of the tissues concomitant with old age. This alteration is general in all parts of the body, and is characterized by a tendency to chronicity following on a lengthened period of development. It is seen in both the reaction to infection, and—what is equally important—in the delay of healing processes. As a clinical illustration of the first, one may cite senile tuberculosis of the cervical glands, and of the second, the greatly increased time necessary to produce adhesion between two layers of peritoneum. It is, indeed, the behaviour of the peritoneum that accounts for many of the anomalous cases of appendicitis under consideration.

Before going more fully into the symptomatology of the clinical types, there are certain features which are common to nearly all cases. There is, first, a much longer history than is usually obtained, and it is not uncommon for seven or eight days to elapse before the symptoms become acute enough



to force the patient to seek surgical aid. This is in marked contrast to the customary twenty-four or forty-eight hour illness with which we are confronted. The average length of history in my cases was between five and six days. There is far less constitutional disturbance, and the amount of pyrexia is unusually small. Pain is comparatively less when due solely to the inflamed appendix, and when marked is the result of secondary peritonitis and distension. All visceral processes are considerably slowed, and constipation is the rule.

The majority of cases fall into a group presenting nearly all the signs and symptoms of acute or subacute intestinal obstruction, rather than an acute inflammatory lesion. There is a history of constipation lasting seven or eight days, the patient passing neither faeces nor flatus. Even more striking in this regard is the failure, often experienced, to produce any relief by repeated enemata, a circumstance strongly suggestive of an organic obstruction. Equally misleading is the history, sometimes given spontaneously by the patient, of increasing difficulty extending over a period of several months.

Vomiting is not a constant feature, but quite often occurs at the onset of the illness, subsiding completely by the time the patient is seen by the surgeon. This is rather in favour of an inflammatory as opposed to an obstructive lesion. The amount of pain varies, but is mostly the outcome of abdominal distension, and its distribution is a wide one, over the whole of the lower half of the abdomen. Not infrequently the pain and tenderness are most strongly marked in the left side, due largely to the upward displacement of the pelvic coils of distended ileum. The abdomen as a whole is markedly distended and resonant. The tongue is dry and furred, and the breath offensive. On rectal examination the bowel is found to be empty, and the frequent failure of enemata to bring relief shows that this emptiness extends far above the portion within reach of the examining finger. I cannot illustrate the condition better than by giving the notes of two actual cases which have come under my care.

*Case 1.*—Mrs. E. M., age 62, was admitted to the West Norfolk and King's Lynn General Hospital on March 15, 1927. On March 8 she began to have daily sickness, but vomited only small quantities. The sickness was associated with some abdominal pain. The bowels had been confined since the first day of the illness—that is, for seven days—and the day previous to admission two enemata had been given without result. On admission two more were given, and the second brought away only a few small fragments of hard faeces. She volunteered that she had noticed increasing constipation for six months. On examination she looked very ill. The tongue was dry and furred. The abdomen was much distended and uniformly tender and resonant over the whole lower half. The temperature varied between 99° and 100°.

A diagnosis of acute intestinal obstruction was made, and the abdomen opened by a mid-line incision. Peritonitis was found, and a gangrenous appendix, lying over the pelvic brim and quite free from adhesions, was removed. The abdomen was drained. Healing was delayed, but the patient recovered and was sent home well at the end of five weeks.

Here was a case with all the history, signs, and symptoms of acute intestinal obstruction in which an error of diagnosis might well be excused.

In the next case to be recorded, one was similarly misled, but fortunately the correct diagnosis was arrived at before operation.

*Case 2.*—Mr. J. S., age 75, was admitted on July 28, 1929. He gave the following history. Increasing constipation for six months without any diarrhoea or passage of blood. For four days previous to his admission he had absolute constipation accompanied by generalized abdominal pain. He had vomited once.

On examination he was a frail wasted old man. The abdomen was moderately distended and slightly tender over the lower half. His temperature never exceeded 99°. Enemata produced only small pieces of faeces and a little flatus. It was decided to operate on him as a case of intestinal obstruction, but on examination of the abdomen under the anaesthetic, a swelling low down in the right side, which could not be felt before, became apparent. A muscle-splitting incision was made, and an appendix abscess drained, the appendix itself being removed. He made an excellent recovery and was discharged at the end of three weeks.

The other cases in this group give very similar histories, but the feature of them all is absolute constipation for a varying number of days, associated with sickness and marked abdominal distension. What we are confronted with in reality is an acute infective process in which the local reaction is so slow and mild that by the time the patient seeks surgical aid the original disorder is masked almost entirely by a new one, which is no more than a complication of it. Doubtless if these cases were seen in the early stages, the difficulties of diagnosis would be far less. That they are not seen is only a reflection of the mildness of the initial symptoms.

The diagnosis of these cases is always difficult, but there are a few points which, borne in mind, may enable us to arrive at the correct conclusion. First of all, although apparently superfluous, I would suggest that acute appendicitis should be considered as a possibility in all cases of acute or subacute intestinal obstruction. Secondly, the distribution of pain and tenderness should not be allowed to influence the diagnosis too much. I have already mentioned that left-sided pain and tenderness sometimes are predominant. I should like to stress this point, as it is of frequent occurrence, and due, as I have repeatedly confirmed at operation, to distended and inflamed coils of ileum which have risen from the pelvis. In old people, therefore, we are faced with this seeming paradox, that in such cases of acute appendicitis the local reaction is comparatively so small that maximum pain and tenderness are left-sided and due to a secondary distension, whereas in cases of true organic obstruction in the large bowel, maximum pain and tenderness may be, and often are, right-sided, owing to the easily distended caecum.

(Since commencing this article, I have been asked by a colleague to see a further case in which this situation confronted us, and in spite of marked left-sided pain and tenderness, and considering the history carefully, a diagnosis of appendicitis was made and confirmed at operation. There was no shutting off of the inflamed appendix, and the pelvic coils of ileum were distended and displaced upwards.)

A third point in the diagnosis is the presence of pyrexia, which, though comparatively little, is definitely in favour of an inflammatory lesion as opposed to a purely obstructive one. Vomiting is less marked than would be expected in an obstruction with the same degree of distension.

No single one of these signs is sufficient evidence on which to make a diagnosis, but by bearing them all in mind, I believe that in nearly all cases one can arrive at the right conclusion.

The intra-abdominal findings at operation are worth recording, as they shed considerable light on the peculiar signs and symptoms. I have already made some reference to the appendix itself. The most striking feature is the freedom from adhesion to surrounding structures and omentum, even after an illness of over a week. It may have a patchy covering of thin lymph, which spreads on to the cæcum and terminal coil of ileum, but there is no complete shutting off, so that a way is clear for the passage of pus and exudate into the pelvis and general peritoneal cavity.

Where peritonitis is present, the pus is often of an unusual nature, being very scanty and extremely greasy, yet forming a thin film over the whole pelvic contents. It is more common to find this condition than an excess of exudate. The pelvic coils of small intestine, as already noted, are injected and distended secondarily to pelvic peritonitis, but occasionally the distension may be partly due to a mechanical obstruction at the ileocecal angle by attempts at forming adhesions in the vicinity of the primary infection. The distension of the ileum in these cases never seems to call for active relief, and subsides quite quickly when the abdomen is drained.

The treatment of these cases is always operative, and the abdomen should be opened by a mid-line incision. Owing to the absence of adhesion, the appendix can nearly always be delivered easily and safely. The peritoneal cavity should always be drained adequately, preferably by corrugated rubber sheeting. If removal of the appendix is difficult, or unwise in view of the patient's general condition, it may be safely left and drainage alone instituted. Although a less desirable procedure, it does not affect the prognosis materially. I have several times been obliged to leave the appendix, but have not lost a patient by so doing.

The prognosis is, on the whole, quite good, *taking into account the age of the patient* and the poor reaction which is so typical of the class under consideration.

While nearly all my cases fitted more or less closely into the group I have described, there have been two others in which the slow reaction was so exaggerated as to present still another picture altogether. Although the condition is rare, I have thought it worth while to record the salient features.

In both cases there was a history of pain, more or less continuous in the right iliac fossa for several months, associated with constipation and occasional diarrhoea. The onset of the illness was mild, and could not be definitely fixed. On examination there was found a mass in the right iliac fossa, which was hard and fixed, but not very tender, and was to all appearances a neoplasm in the region of the ascending colon. The first was, after investigation, operated upon as such, but an appendix abscess was found. The contents were odourless and composed chiefly of mucus and were probably sterile. In the second case, between first seeing the patient and admitting her to hospital, a period of three weeks elapsed, and in that time the swelling, which had been very marked, almost completely disappeared. In her

case it was found at operation that only a very small quantity of pus remained, and that the abscess had practically resolved spontaneously.

Here again, arrival at the correct diagnosis depends largely on having in mind the possibilities of an extremely chronic inflammatory condition in the appendix. The absence of occult blood in the fæces is strong confirmation of such a state of affairs.

It is quite safe to temporize with these cases, as by postponing operation resolution may occur; but it is wise to remove the appendix at a later date, as it nearly always gives rise to symptoms and may at any time flare up again.

### SUMMARY.

1. Consideration is given to anomalous forms of appendicitis in old people.
2. Their similarity to cases of acute obstruction is noted.
3. The symptoms are reconciled with the pathological findings.
4. The treatment is indicated.

## A CONTRIBUTION TO THE SURGERY OF THE PITUITARY REGION.

### AN ACCOUNT OF FOUR CASES OF 'PITUITARY TUMOUR' TREATED BY RADON SEEDS.

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OWING to the recent death, and autopsy, of the third case of this series, the time has arrived when the four cases can be reported, all operated on during the last six months of the year 1929. They form, I believe, the first series of 'pituitary tumour' cases in which an attempt has been made to improve the condition of the patient by the introduction of radon seeds into the tumour mass. This short contribution to the surgery of the pituitary region deals solely with the technique, etc., of radon-seed introduction, and the results obtained, together with some comments.

In the experience of surgeons interested in the surgery of the pituitary region, it would appear that: (1) 'Pituitary tumours', in their various phases and aspects, are on the increase; (2) Their recognition is more readily made than heretofore; and (3) The ordinary surgical procedures are associated with a very undesirable mortality-rate and also unsatisfactory in their end-results. Consequently any new procedure merits consideration.

The introduction of a radon-seed manufactory at St. Bartholomew's Hospital suggested the possibility of a trial in respect of 'pituitary tumours', and a case soon presented itself that fully justified the attempt (*Case 1*). The result obtained was sufficiently encouraging, and three other cases were treated in the same manner in the same year. The four cases not only demonstrate the feasibility of radon-seed introduction, but also show that some benefit results—for example, the first case so treated was obviously dying when in hospital, and yet lived nine months in comparative comfort; whilst the fourth case, now living in Ireland, has benefited very greatly. It is impossible from this number of cases to claim more than this, and it must be open to question whether the improvement observed in these two cases could not be accounted for by the general decompressive action of the operation. Further experience is needed before this question can be settled.

It is necessary, as a preliminary, to be quite clear as to the justification of any major procedure, whether the ordinary attempt at tumour exposure followed by partial extirpation by sucking or electro-surgery, the introduction of radon seeds, or any other similar measure, and the indications as laid down by Sir Percy Sargent may be taken as a sound guide—for the relief of intolerable headache, and for the saving of the eyesight when the tumour

# SURGERY OF THE PITUITARY REGION 69

is progressing in the forward direction and threatening the optic nerves and the optic chiasma.

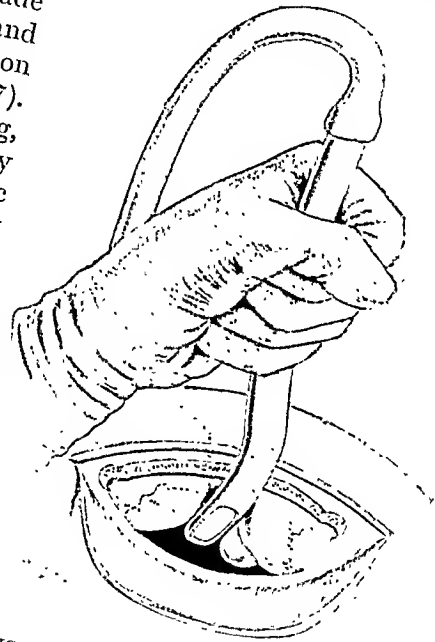
In all four cases the same method of seed introduction was carried out—a lateral frontal bone-flap (right in three cases, left in one), the mesial margin of the flap falling short of the middle line, thus avoiding the superior longitudinal sinus, and the lower border of the flap passing close above the supra-orbital margin—the flap turned outwards. If the frontal sinus is exposed, the cavity is sterilized and packed with gauze till the termination of the operation.

The dura mater enclosing the frontal lobe is raised from the orbital plate as far back as the posterior border of the anterior fossa, and the dura and brain are retracted with the aid of a special curved metal retractor with light attached—the pattern illustrated was made for me by Messrs. Allen & Hanburys, and it forms, I think, a distinct advance on other illuminating retractors (*Fig. 37*). The wires are enclosed in rubber tubing, and the whole can be sterilized by immersion in lysol, etc. The wires are connected with a small dry-cell battery concealed in a pocket in the operating gown.

The basal region of the anterior clinoid process is verified, and a small incision made in the dura, as near to the base of the process as possible. Through this slit a blunt director is passed, directed backwards, inwards, and downwards, till it strikes the bone at the base of the pituitary fossa. Whether the tumour is of the body itself or of the suprasellar region is estimated beforehand; this will naturally influence one in the direction of the guide.

Previous experimentation on the cadaver showed that a blunt instrument could be thus introduced into the pituitary fossa without material damage to the various important anatomical structures related thereto. The guide passes above and internal to the cavernous sinus and the structures in relation to it, anterior to the front part of the circle of Willis, and with but slight damage to the brain (temporo-sphenoidal pole). Neither the brain of *Case 2* (in the Museum of St. Bartholomew's Hospital) nor that of *Case 3* (in the Museum of the West End Hospital for Nervous Diseases) shows other than the slightest degree of cerebral lesion. The second case certainly died shortly after the operation, but the small amount of blood effused at the base of the brain was derived from a vessel of the tumour itself, bursting probably as the result of the general decompression.

Along the track of the director the special seed-introducer is passed, and



*Fig. 37.*—The illuminating retractor.

the seeds are expelled—spaced as far as circumstances permit, by lengthening or shortening the introducer and by alteration of its angle to the base of the skull. That this spacing can be carried out is proved in all four cases, as seen on the X-ray pictures (*see especially Figs. 38, 39*).

**Number of Seeds Introduced.**—Fourteen seeds in *Case 1*, 9 in *Case 2*, 8 in *Case 3*, and 8 in *Case 4*, each seed representing 1 millicurie radon emanation. The only guide to the number of seeds requisite for the case is the degree of bone destruction, as representing the size of the tumour mass, assisted perhaps by the intensity of the symptoms. The two autopsies showed, however, quite convincingly, that many more seeds were required to include the main mass of the tumour than were actually utilized. This is one of the lessons learnt from the four cases. On the other hand, the surgeon having introduced a certain number of seeds with safety to the patient, naturally hesitates to insert the greater number.

In all four cases the diagnosis was clear, two sellar tumours and two suprasellar, and no attempt was made at the operation either to see the tumour or ascertain its dimensions. It is possible that this method of 'blind insertion' may be criticized, but it should be noted, in defence, that the exposure of the tumour adds materially to the danger of the operation, and that, in any case, it is usually quite impossible to determine the size, shape, and extent of the tumour mass.

Further points in respect of the number of seeds used, their spacing, etc., will be brought out in the description of the four cases.

### CASE REPORTS.

*Case 1.*—Admitted into St. Bartholomew's Hospital under the care of Sir Thomas Horder, suffering from 'blindness'. A blow had been received over the left frontal region twelve weeks previously, and subsequently the patient had suffered from ever-increasing headaches and loss of sight. There was marked proptosis of both eyes.

**ON EXAMINATION.**—Without going into unnecessary clinical details, the neurological report was to the effect that the neoplasm was not exactly of the pituitary substance but rather of the base of the skull, invading the base in the downward direction, and upwards into the substance of the base of the brain in the immediate neighbourhood of the pituitary fossa. The X-ray report stated that "the sphenoidal sinus appears clear. The pituitary fossa is not recognized, and the whole of this part of the sphenoid, including both anterior and posterior clinoid processes, has disappeared. The appearances are consistent with a tumour eroding the pituitary fossa and encroaching upon the sphenoid, suggesting malignancy."

**OPERATION** (Oct. 10, 1929).—After consultation with Sir Thomas Horder and Dr. Finzi, I determined to see whether radon seeds would do any good. The patient was much worse, very drowsy, with increased bulging of both eyes, incontinence of urine and feces, and at times quite unconscious. During the operation periodic blood-pressure tracings were taken, to act as a guide as to whether the introduction of the seeds could be completed or not. The following are the records obtained :—

At the beginning of the operation	..	140/90
On the turning back of the bone-flap	..	140/90
On lifting up dura and brain	..	120/80
On introducing the seeds	..	130/90
At the termination of the operation	..	190/85

The patient was X-rayed the following day, the report being "fourteen seeds can be identified in the middle line, two-thirds just anterior to the pituitary fossa,

and six lateral, three on either side." The accuracy of the insertion and the spacing was encouraging (*Figs. 38, 39*).

**SUBSEQUENT HISTORY.**—For two or three days after the operation, the patient was worse and expected to die, but on or about Oct. 20 he improved to a marked



**FIGS. 38, 39.**—*Case 1.* Lateral and antero-posterior views of the skull. Fourteen seeds situated in the pituitary region. The pituitary fossa and its osseous boundaries are destroyed by growth. The seeds are accurately placed and well spaced.

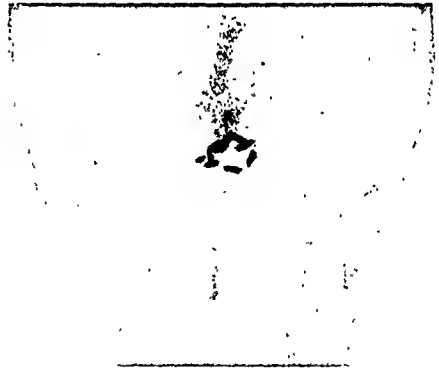
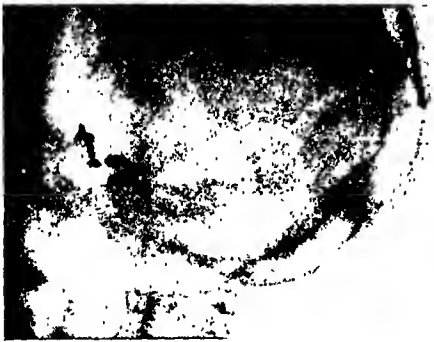


degree, talked rationally, and the bulging of the eyes was much less obvious. Subsequently his condition improved to such an extent that he was discharged to his home in the Midlands. There further improvement took place: the headache was relieved and the general condition became so much better that he was enabled to go to local football matches, on which game he was an enthusiast. Six months after the operation he fell dead in the street. There was no post-mortem examination.

**Comment.**—Whether the improvement that took place after the operation was due to the effect of the seeds or the decompression is open to question, but the feasibility of seed introduction was demonstrated, and it was also clear that the seeds could be inserted with fair accuracy and spacing. These facts encouraged me to try again.

**Case 2.**—A young girl, age 13, was admitted to hospital under the care of Mr. Foster Moore, in September, 1929, suffering from severe headache and loss of vision. The symptoms commenced in May of the same year. The headaches became ever more severe, mainly frontal in position, and progressive eye trouble induced her to seek advice.

**ON EXAMINATION.**—It was found that the girl was suffering from marked papilloedema in both eyes, 2 to 3D. This steadily increased and was followed by paralysis of the left sixth nerve. The visual fields showed well-marked bitemporal hemianopia. The X-ray report stated that "the pituitary fossa is definitely



FIGS. 40, 41.—Case 2. Lateral and antero-posterior views of the skull, showing nine seeds, accurately placed and well spaced.

enlarged for a child of this age, with apparent erosion of the posterior clinoid process—also abnormal shadows above the fossa, the appearances strongly suggestive of a pituitary tumour" (the tumour proved to be a suprasellar adamantinoma).

**OPERATION (Nov. 20).**—Nine radon seeds were inserted, without any difficulty, as in the previous case. I felt that the seeds had been inserted into a tumour mass, as definite resistance was encountered by the introducing instrument. The patient left the operating theatre in excellent condition, but during the night respiration, pulse-rate, and blood-pressure all showed a high grade of intracranial pressure, and the patient died.

**AUTOPSY FINDINGS.**—In view of the importance of the case, X-rays were taken shortly after death (Figs. 40, 41). At the autopsy the brain was removed and hardened, and subsequently X-rayed. A large suprasellar tumour was seen, with all nine seeds buried in the tumour, seven in the ipsilateral half of the tumour and two in the middle line (Figs. 42-44).

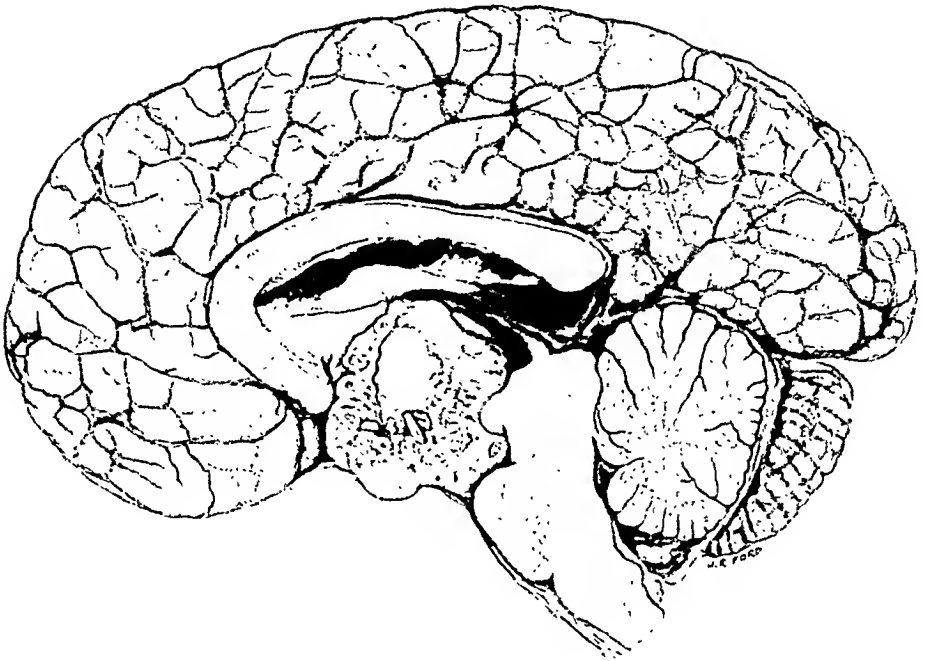


FIG. 42.—Case 2. Two seeds are seen in the centre of the tumour in the middle line. (Drawn from the specimen in the Museum of St. Bartholomew's Hospital.)

**Comment.**—This case again demonstrated conclusively that the seeds could be implanted accurately. The cause of death was not quite clear. There was some hæmorrhage at the base of the

FIGS. 43, 44.—Case 2. Right and left halves of the brain. A wire circle surrounds the tumour mass, and it can be seen that the contralateral half of the tumour (on the side opposite to the bone-flap) contains no seeds, but that all the seeds introduced (eight in number) are situated in the tumour substance on the ipsilateral side. The ventricle is markedly dilated.

brain, but the source of this bleeding could not be determined; it looked as if some vessel of the tumour itself had burst as the result of the general decompression.



*Case 3.*—A boy, age 8, was admitted to the West End Hospital for Nervous Diseases, under the care of Dr. C. C. Worster-Drought. He had suffered from severe headaches with vomiting for the previous ten months. The sickness came on in the morning, followed by headache, mainly frontal in site, and then screaming attacks. There were intervals in which he appeared to be better, but he became progressively worse. Sometimes he suffered from vertigo, falling always to the right. He was very irritable, emotional, and often hysterical.

#### ON EXAMINATION.—

It was found that he had marked papilloedema in both eyes, equal to 4D. Fields normal except perhaps for a slight narrowing in the temporal fields. Headache more or less constant and vomiting of the cerebral type.

July 12.—The patient was very drowsy all day. The X-ray picture showed enlargement of the pituitary fossa and calcification immediately above the sella, suggestive of the calcification of an adamantinoma of the suprasellar region.

OPERATION (July 17).—Eight radon seeds were inserted, without any difficulty.

July 19.—He seemed much better and stated that he could see better.

July 27.—Improvement maintained. Considerable bulging in the region of the bone-flap. Papilloedema 2D.

Aug. 16.—Much better, little headache, and mentality much improved.

Sept. 10.—Discharged from hospital.

RE-ADMISSION.—The boy was re-admitted on Nov. 30. His progress since discharge could be summarized as follows: First week: not well, headaches. Second week: very well. Third week: severe headaches, vomiting, and screaming attacks.

Subsequently there was some improvement, he smiled at times, and answered questions. Pupils dilated and dises white. Owing to the increasing forward bulging of the bone-flap, the flap was turned down and the bone removed.

FIGS. 45, 46.—*Case 3.* Lateral and antero-posterior view of the skull. Seeds well spaced, but more widely spread than in the previous cases.

The lad remained in hospital during the whole of the year 1930, steadily going downhill. Owing to the progressive bulging at the wound, various efforts were made to reduce this, both by lumbar puncture and intravenous injection of hypertonic saline solution. For example, on Dec. 4, the following was noted after lumbar puncture. Withdrawal of 5 c.c. cerebrospinal fluid—headache relieved; 10 c.c.—flap bulging reduced; 20 c.c.—further reduction; 30 c.c.—severe headache.

Dec. 5.—Injection of 50 c.c. of a 30 per cent hypertonic saline solution was followed immediately by increased bulging of the flap, marked headache, and vomiting.

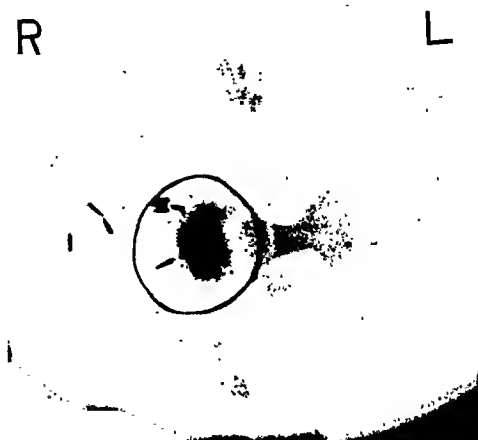
Dec. 6.—The same measure was repeated with even more severe results, these being at once alleviated by the withdrawal of 40 c.c. of cerebrospinal fluid.

Dec. 15.—Intravenous injection of 50 c.c. of a 15 per cent hypertonic solution. At 45 c.c. there was no change, but at 50 c.c. slight headache and some increased bulging of the flap.

The X-ray picture showed that the eight seeds were arranged as follows: Three in the fossa, three in relation to the anterior clinoid process, and two in relation to the posterior (*Figs. 45, 46*). The lad died early in 1931.

**AUTOPSY FINDINGS.**—At the post-mortem examination a large suprasellar tumour was found, calcified at its base and extending into the ventricular area, the ventricles themselves being markedly dilated, more especially towards the area which had been decompressed.

The brain was X-rayed. Difficulty was experienced in arranging the brain in a natural position; it was very soft, and the forward bulging of the left frontal pole added to the difficulties. The picture showed, however, that five seeds were concealed in the tumour mass, within the wire circle, but that three had wandered laterally and looked to be lying free in the dilated ventricle (*Fig. 47*). The specimen is being hardened and examined further.



**FIG. 47.**—*Case 3.* Skingram of the brain, showing five seeds within the wire of the tumour circle, the great dilatation of the ventricles, more especially in the forward direction on the side operated on, and three seeds apparently extruded into the contralateral ventricle.

**Case 4.**—A man, age 31, was admitted to the West End Hospital for Nervous Diseases under the care of Dr. Worster-Drought. A year and a half previously, when in Kenya, he was operated on for persistent headache—subtemporal decompression, with at first considerable improvement. Subsequently, the headaches returned; he suffered from giddiness and impaired vision.

**ON EXAMINATION.**—It was found that the patient had papilloedema in both eyes, 1 to 2D. Visual fields normal. X-ray showed marked sellar enlargement and definite local destruction of bone.

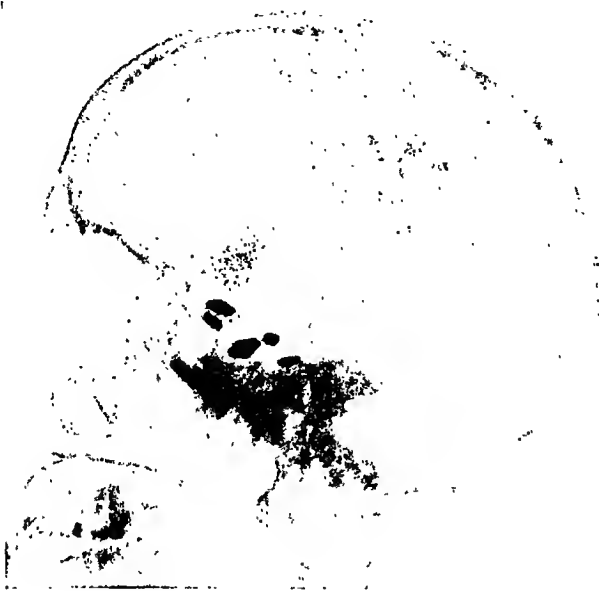
**OPERATION (Nov. 11).**—Eight seeds were inserted, without difficulty. Subsequent to the operation there was marked improvement in all respects. X rays showed that the seeds were in fair position (*Figs. 48, 49*).

**SUBSEQUENT HISTORY.**—The after-history of this patient is interesting. In the first place, early in December, he developed marked polydipsia and polyuria. Up to the end of December the intake averaged about eighteen pints during the twenty-four hours, with corresponding output. In all other respects he felt well. He was then discharged.

The present condition of this patient, from a letter received (from Ireland) in December, 1930, more than one year after the operation, runs as follows: "My general health is good, my sleep is only fair. My weight when entering the hospital was 9 st. 2 lb. My weight is now 11 stone. My vision seems to be somewhat better, and I think the glasses have helped them. My thirst and abnormal passing of water has now returned to normal. My gait is very much better but not yet perfect. I am a good deal steadier. I am doing a good deal in the line of exercise and did a good deal of swimming in the summer. I walk on an average

nine miles a day. In every way I feel very much better—hearing, speech, strength, everything I can think of.”

The eye-report reads as follows: “Full fields. Nystagmus on looking to the right, also present but less marked on looking to the left. Pupils equal in size, each



FIGS. 48, 49.—Case 4. Lateral and antero-posterior view of the skull, showing eight seeds in the pituitary region, well spaced, though two seeds are rather lateral. The decompressed region of the skull (operation in Kenya) is seen in both figures.

reacts to light and convergence. The discs are lacking in sharpness of outline, and there is, I think, slight narrowing of vessels, especially in the left eye—appearance is that seen after papilloedema that has been relieved by decompression.”

**Comment.**—Altogether this case may be regarded as highly satisfactory. In respect of the polydipsia, etc., I have observed this phenomenon on a previous occasion. A patient was admitted to St. Bartholomew's Hospital under my care suffering from a fracture of the middle fossa of the skull. There was bleeding from both ears, and it was clear, from researches carried out by me many years ago, that the fracture passed through the body of the sphenoid. This patient developed polydipsia, etc., within a day or two of admission to hospital, and this continued for the next ten days or so, gradually returning to the normal and ceasing in about three weeks. It was considered at the time in this case that the pituitary body had been injured, and in *Case 4* it seemed clear that the introducer or the seeds had brought about a similar change in the pituitary function.

### CONCLUSIONS.

From a general résumé of these four cases, it is clear :—

1. That radon seeds can be introduced into and spaced throughout a tumour of the pituitary region.
2. That the neighbouring structures can be avoided.
3. That the results obtained are not discouraging.

## TRAUMATIC ARREST OF EPIPHYSIAL GROWTH AT THE LOWER END OF THE TIBIA.

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AND THE DAVID LEWIS NORTHERN HOSPITAL.

It has always seemed interesting that while a very small proportion of injuries to the lower end of the radius during the period of epiphysial growth are followed by a progressive deformity, the reverse is true of the tibia. After trivial trauma at the ankle a deformity giving rise to serious disability may arise. The proportionate rarity of similar trouble in the wrist is due to the difference in the shape of the joint and in the nature of the injury. There



FIG. 50.—The type of fracture which gives rise to deformity similar to that shown in Fig. 02.



FIG. 51.—Injury sustained seven years previously. This man did full work as a mechanic.

is no 'mortice' at the wrist-joint, movements are free, and a fall usually displaces the epiphysis and growing cartilage completely with a slice off the diaphysis. Only when the epiphysis is crushed does arrest of growth occur (Fig. 50). The deformity which then ensues consists of radial deviation and prominence of the lower end of the ulna. Possibly some of these cases are

classed as Madelung's deformity. There is very little disability. In one case the man earned full wages as a mechanic (*Fig. 51*).

Occasionally the lower tibial epiphysis is bodily displaced (as is usual at the wrist), and it can then be shown that there is no subsequent interference with growth.

On the other hand, at the ankle the astragalus is firmly held by the tibial and fibular malleoli. Lateral movement is not free, and if it is forced, an undue strain is put upon the malleoli. Where children are concerned, this strain falls on epiphyses, and it is found that the injury preceding tibial arrest almost always consists of an inward twist of the foot. The astragalus is forcibly adducted, the inner edge of the upper sur-

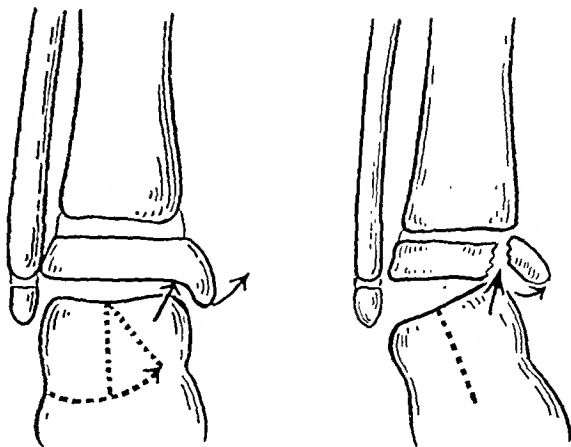


FIG. 52.—Mechanism of epiphyseal fracture.

face impinges on the inner half of the epiphyseal plate of the tibia and applies a crushing force at this point (*Fig. 52*). At the same time the internal malleolus is forced upwards and inwards and the epiphysis breaks at the base of the malleolus.

It was interesting to hear from Willis Campbell that this epiphyseal fracture is, in his opinion, rare in the United States. In this country cases occur fairly frequently, and there are 23 cases in my series (two years—about 3000 fractures). For some time this discrepancy seemed inexplicable, but the solution probably lies in the causative agent. This is in nearly all cases a railing composed of regularly spaced vertical iron rods used to enclose gardens and parks; the children are addicted to climbing over them and slip with one foot jammed between the two rods. Such railings abound in England, and are apparently extremely scarce in America. The constancy of the history is well illustrated by the following incident:—

I was asked to see a boy as an out-patient who had hurt his leg the previous day, and on my way was shown the skiagram (*Fig. 53*); this showed that he had injured his epiphysis about a year ago. I informed the house surgeon that in all probability his recent injury could not be respon-



FIG. 53.—Injury one year previously.

sible for this X ray. Before the patient was examined his older brother was asked if about a year ago he was climbing some railings and slipped with his foot caught fast. The brother immediately remembered the incident



as he had had to free the boy and carry him home. The boy had limped for a week or so, had then apparently recovered, and the whole thing had been dismissed as a sprain.

This is the typical history. The disability is transient and slight at the time. After a year a deformity may be noticed and is seen gradually to increase, the attention of the parent being drawn to the 'going out' at the ankle and the uneven wearing away of the boots.

It will be seen from the skiagrams that, following the crushing of the inner half of the epiphysial disc of the tibia, this half only ceases to grow, presumably owing to the death of cartilage cells, either from being crushed or from hæmorrhage into them. The outer half and the fibula continue to grow at the normal rate, and the resulting deformity is what would be expected. The ankle-joint turns inwards just as surely as a door swings on its hinges.

Thus a varus deformity of the ankle develops owing to a low outward bowing of the tibia and fibula. The external malleolus may be as much as  $2\frac{1}{2}$  in. below the level of the internal. The astragalus goes with the malleoli,

and its vertical deviation is incompletely corrected at the mid-tarsal and talo-calcaneal joints.

The treatment of the acquired deformity is simple and efficacious. Through an external incision the fibula is exposed just above the ankle-joint. A wedge consisting of fibula and three-quarters of the thickness of the tibia is removed, as in *Fig. 54*. The line of this passes through the interosseous ligament where the two bones are practically

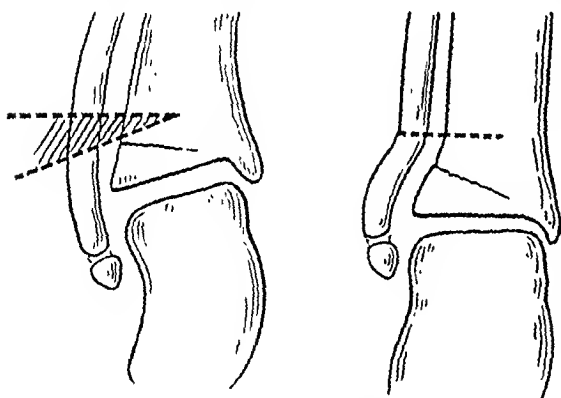


FIG. 54.—Diagram showing the method of operative correction of the deformity.

in contact. The upper surface of the wedge should be horizontal, the lower parallel with the lower tibial articular surface.

Unless the patients are importunate, operation should not be done in cases under 14 years of age, and it is best to wait until 18 to 20 years, when epiphysial growth has ceased. One case under 14 was corrected, and in order to minimize the shortening of the leg a piece was taken from the fibula on the outer side, the tibia was divided from the inner side, deformity was corrected, and the piece of bone was put into the wedge-shaped gap in the tibia.

Attempts have been made, as one would expect, to prevent the occurrence or progression of the deformity. These have not been definitely successful.

The following skiagrams (*Figs. 55–64*) are a selection from a large number of cases illustrating the constancy both of the type of injury and of its resulting progressive deformity.



FIG. 55.—Twisted foot in railings a month previous to photograph.



FIG. 56.—Same case as Fig. 55, seven months after accident.



FIG. 57.—Same case as Fig. 55, two years after accident.



FIG. 58.—At the age of 10 this patient was climbing railings when he fell, and one foot stuck. Nine years after accident.



FIG. 59.—Same case as Fig. 58. Corrected January, 1929. Photograph five months later.

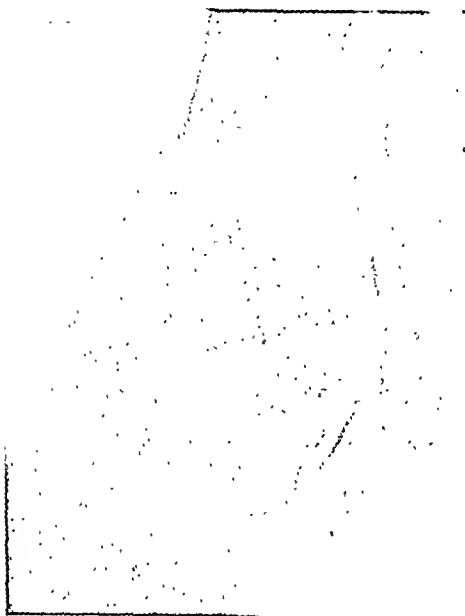


FIG. 60.—A case successfully treated by osteotomy. Fifteen years after accident.



FIG. 61.—The patient twisted his foot in a grid. Photograph a few days after accident.



FIG. 62.



FIG. 63.



FIG. 64.

FIGS. 62-64.—Same case as Fig. 61. Fig. 62 shows the condition two years after accident. Fig. 63, four years after accident: parents anxious for correction: osteotomy in June, 1928. Fig. 64, one year after correction by cuneiform osteotomy of fibula and insertion of the piece of bone into the gap of a linear osteotomy of the tibia.

**CARCINOMA OF THE THYROID GLAND.**

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*(Being an Arris and Gale Lecture delivered at the Royal College of Surgeons of England on February 4, 1931.)*

MORE than ordinary interest attaches to carcinoma of the thyroid gland because it has been stated that it has features which are not known to occur in malignant disease of any other organ. Some of these, if true, would destroy the basis on which our ideas of malignancy are founded. It has been asserted that a benign tumour of the thyroid, and even a normal gland, may give rise to metastases. This belief is associated with names distinguished in surgery and pathology such as Lucke, Cohnheim, von Eiselsberg, Kraske, and others. It is now generally agreed that a secondary growth may reproduce some of the morphological characters of the primary growth, but it has been stated that the thyroid malignancies do more than this in that the metastases may be indistinguishable from the normal gland tissue. A malignant growth should have no physiological activity, yet von Eiselsberg has shown that a metastasis from a malignant thyroid tumour can function to an extent adequate for the body's needs after extirpation of the parent gland, and thyroid carcinoma tissue has been proved to have functional activity by the tadpole test.

The case reports upon which some of the above assertions were founded have been critically examined by Bérard and Dunet,<sup>1</sup> Joll,<sup>2</sup> Graham,<sup>3</sup> and others, and reviewed in the light of their own experiences. It is clear from their results, and from our own investigations, that some of the apparently anomalous features of the pathology of the thyroid are to be attributed to the fact that the character of the cells and their arrangement vary so greatly in growths that are benign. The extent of this variation may be such that it becomes difficult to determine when the activities of a growth have crossed the line dividing innocency from malignancy. For much of the recent work devoted to this aspect of the subject we are indebted to Allen Graham,<sup>3</sup> at Cleveland; Simpson,<sup>4</sup> at Ann Arbor; Asehoff,<sup>5</sup> Wegelin,<sup>6, 7</sup> and Bérard<sup>8</sup>, on the Continent; Scott Williamson and Innes Pearse,<sup>9</sup> in London; and Tebbutt,<sup>10</sup> in Sydney.

The purpose of this paper is not to cover the whole field of carcinoma of the thyroid, but rather to present those observations which have been made on the clinical and pathological material occurring in my work. This material has been gathered while working in the Surgical Professorial Unit of St. Bartholomew's Hospital, and before that in Australia. It is inevitable in clinical investigation that gaps in our inquiries and observations on individual patients should occur, and therefore finality is not claimed for the

views presented. The diversity of the histological picture adds a peculiar difficulty to the investigation of the pathology of the thyroid gland, and I am indebted to my colleagues of the pathological department for their assistance.

**Classification.**—The classification of carcinoma of the thyroid varies in every country, and with every author I have read. Many classifications are long and confusing. The reason for this is that comparatively few workers have directly concerned themselves with this problem. For the most part each has worked more or less alone and has been intent upon the material of his own clinic, and each has evolved a classification which seemed best to meet his needs. Nevertheless uniformity is beginning to appear.

The classification I have adopted is similar to that published by Allen Graham.<sup>3</sup> It is adequate, and there is neither necessity nor reason for the more complicated subdivisions adopted by many. This classification divides the carcinomata into : (1) Scirrhus (or carcinoma simplex) ; (2) Papilliferous adenocarcinoma ; (3) Malignant adenoma.

Objections can be raised against this classification in that the second and third groups tend to overlap. This overlap occurs because, whatever the stimulus to cell multiplication, or the type of carcinoma induced, thyroid epithelium retains to some extent a capacity to produce any of its morphological characteristics—that is, the cells may be arranged in anastomosing columns, or in follicles, or the structure may become papilliferous. Notwithstanding this, each type has features sufficiently outstanding to place it in its class.

**Criteria of Malignancy.**—The question will continually arise : On what criteria is the diagnosis of malignancy based ? If we are satisfied with the descriptions usual in surgical text-books—which almost invariably refer to the later stages of the disease—no difficulty arises, but by that time the information is useless to the patient, and practically devoid of interest to the investigator. If we are to attempt to recognize cases of malignancy at an early stage—and surely this is our most urgent need—difficulty is met at once. The chief reason for this is that, apart from scirrhus (carcinoma simplex), carcinoma rarely arises in a normal gland ; it almost always occurs in a gland which is already pathological and in which the epithelium, although benign, may show extremely diverse types of structure (*see Figs. 90, 91*), and the change from a benign to a malignant nature may take place with scarcely any change in the character or arrangement of the cells. When the morphological appearances may be so similar in a malignant and a non-malignant tumour, the criteria of malignancy must sometimes rest, not on the character or arrangement of the epithelial cells, but pathologically on such evidence as invasion of the capsule and invasion of vessels, and clinically on local recurrence or metastases. In both papilliferous adenocarcinoma and malignant adenoma the ultimate criteria are the same, but the difference in the characteristics of the two types is such that each presents its own difficulty in drawing the line between innocency and malignancy, and in each case epithelial activity has been present long before a pathologist would give a definite opinion that malignant transformation had occurred. It may be right for a pathologist to be conservative and refuse to accept a diagnosis until the

proof is beyond doubt, but the welfare of the patient demands that the biological side of the question should be given adequate consideration.

#### Origin of Thyroid Carcinomata.—

1. *Scirrhus*.—Scirrhus (carcinoma simplex) originates in the same manner as scirrhus of the breast. The epithelium breaks through the basement membrane of the alveoli.

2. *Papilliferous Adenocarcinoma*.—From our own observations we know that an innocent papilloma (or one not proved to be malignant) has originated in an adenoma. In some of our cases of papilliferous adenocarcinoma I have not been able to obtain a history of a preceding tumour. Also, I have found papilliferous growth, apart altogether from tumour formation, almost as extensive and active as that found in a tumour (*see Fig. 68*). Again, as will be seen later, in our aberrant thyroids papilliferous change had been present throughout all the nodules and the parent gland. Therefore I believe that papilliferous growth can occur apart from adenoma. Wegelin states: "It is certain that the development of the papilloma follows quite independently of the earlier presence of an endemic goitre. The tumours investigated in the Berne Institute are derived for the most part not from the indigenous inhabitants, but from foreigners who have come from goitre-free regions."<sup>6</sup> (Wegelin employs the term 'papilloma', but states that six of his seven cases were malignant; as used by him, therefore, the term practically corresponds to papilliferous adenocarcinoma and not to the benign neoplasm.)

3. *Malignant Adenoma*.—Malignant adenoma invariably originates in a pre-existing adenoma, and in most published records this type constitutes from 85 to 90 per cent of the cases of carcinoma of the thyroid.

**Incidence of the Types of Carcinoma.**—Scirrhus is comparatively rare. As the percentage of cases of carcinoma arising in a pre-existing adenoma is so high, it will be obvious that the incidence of carcinoma should be higher in those regions where the incidence of endemic goitre with the formation of adenomata is high. Bérard and Dunet<sup>8</sup> conclude that in goitre areas from 2.5 to 4 per cent of all malignant tumours affect the thyroid gland, whereas in goitre-free areas the percentage is only 0.4 to 0.5 per cent. Wegelin<sup>7</sup> states that "these figures speak with no uncertain voice. Where goitre is endemic, malignant tumours of the thyroid occur in greater numbers". In non-goitrous regions I believe that the proportion of papilliferous adenocarcinoma to malignant adenoma is higher, and for that reason the number of cases of papilliferous adenocarcinoma compared with that of malignant adenoma is decidedly higher in my series than in one from a centre of endemic goitre.

Wegelin<sup>6</sup> writes the section on thyroid disease in the *Handbook of Special Pathology* by Henke and Lubarsch. He has an immense amount of material, and he publishes each of his cases of papilloma in detail. There are only seven of these, while it is obvious that his total number of cases of carcinoma is very high. Although, as already stated, he calls these 'papillomata', he shows that six of these have undergone malignant change, and the seventh is a toxic goitre. In our series there have been 9 cases of adenopapilliferous carcinoma in a total of 38 cases.

After excluding doubtful cases, there remain in our series 38 cases, in 32 of which the histories and pathological material are available for study.

This number has occurred in 2127 operations for all types of goitre—that is, 1·78 per cent—but if toxic goitre is excluded it has occurred in 937 patients, being 4 per cent. The latter figure cannot be taken as the average percentage of malignant to non-malignant tumours, because we have been prevented by limitation of beds from admitting any but the more serious cases, or those in which a serious condition has been suspected.

### SCIRRHOUS CARCINOMA (CARCINOMA SIMPLEX).

I do not propose to discuss this type at length. As already stated, it is rare in comparison with the other two types. It does not differ from a similar



FIG. 65.—Scirrhou carcinoma (carcinoma simplex).

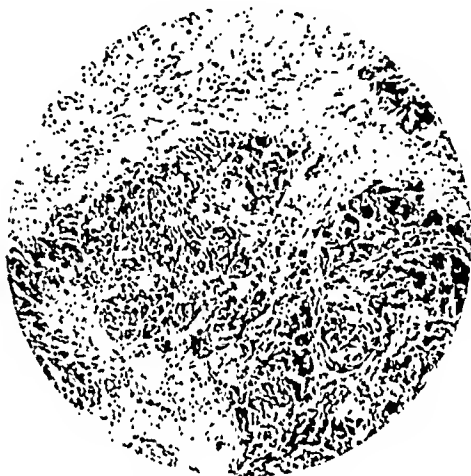


FIG. 66.—Scirrhou carcinoma (carcinoma simplex).

type of tumour elsewhere in the body. The tumour is comparatively small, and very hard. It invades locally, and disseminates to adjacent lymph-nodes. The histopathology is shown in *Figs. 65, 66.*

### PAPILLIFEROUS ADENOCARCINOMA.

In our series this type constitutes 23·6 per cent of the total number.

The distinctive feature of this variety of carcinoma is the papilliferous tendency. Papilliferous structure in the thyroid gland is not confined to malignant growths. It occurs in adolescent goitre, toxic goitre, and benign papilloma, as well as in papilliferous adenocarcinoma, and it is almost always present in lateral aberrant thyroids by the time they are recognized clinically. Marine and Lenhart<sup>11</sup> have readily produced goitre in brook trout, and, in some of these, tumours occur of 'arborescent papillomatous' type. It is scarcely likely that this type of epithelial hyperplasia is of different significance in each of these conditions. It rather suggests that stimulation, whether physiological or pathological, elicits this type of response because of the inherent properties of thyroid epithelium.

The papilliferous response of thyroid epithelium in (1) toxic goitre, (2) benign papilloma, (3) papilliferous adenocarcinoma, (4) aberrant thyroid gland tissue, is illustrated by *Figs. 67-89*.

### 1. TOXIC GOITRE.

*Fig. 67* shows a condition which is frequently seen in toxic goitre. *Fig. 68* is also from a patient suffering from toxic goitre. The extent of



*FIG. 67.*—Papilliferous formation as frequently seen in toxic goitre.



*FIG. 68.*—Papilliferous formation in toxic goitre. The extent of the condition in this case is unusual.

the papilliferous formation is altogether exceptional for this condition. This patient—a young woman—had been ill for twelve months, and after four months in bed was becoming progressively worse.

### 2. PAPILOMA (BENIGN).

This tumour is classed among the benign neoplasms. In my last 1044 operations in which every specimen removed has been examined microscopically I have found only two cases of simple papilloma, and I cannot be sure that one of these is innocent. The condition, therefore, is rare. In one of these (*Fig. 69*) transformation from vesicles containing colloid to papilliferous structure can be seen taking place along a wedge-shaped front. It is obviously an active process. If an area next to the capsule is examined, no invasion is found (*Fig. 70*). This must be regarded as benign, but active papilliferous change is taking place, and I do not know where (or whether) that activity will stop. Wegelin states that "one cannot say with certainty where the normal tissue ends and the tumour tissue begins."<sup>6</sup> In the next patient the condition had advanced a stage further (*Fig. 71*). We see a similar condition throughout the nodule, but examination immediately beneath the capsule shows great irregularity in the size and shape of the cells, and also what I believe to be invasion of the capsule (*Fig. 72*).



These two points, irregularity of shape and size of cells, and invasion of capsule, are capable of two explanations. The inequality of the cells may be due to degeneration, and, instead of the papilliferous growth invading the capsule, the fibrous tissue may be invading and strangling the growth, but the nuclei of the cells stain so deeply that I think they are not degenerating, and probably the growth is invading the capsule. These two tumours are classed

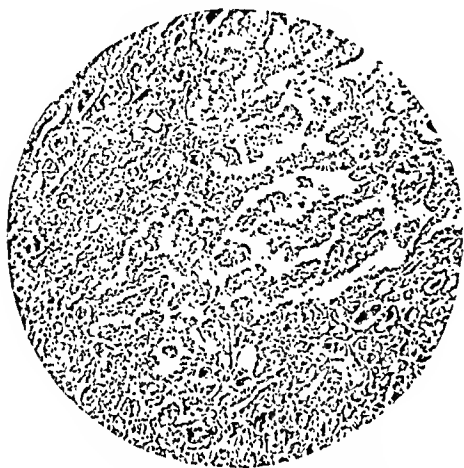


FIG. 69.—A wedge of papilliferous tissue is seen surrounded by vesicles containing colloid.



FIG. 70.—The same case as *Fig. 69*. No invasion of the capsule is found.

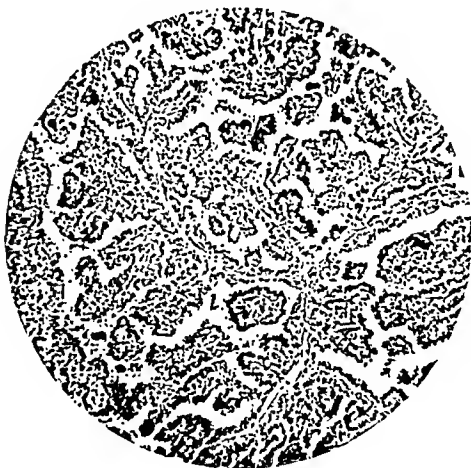


FIG. 71.—Papilliferous change throughout the nodule.



FIG. 72.—The same case as *Fig. 71*. The epithelium has become more embryonic in type, and invasion of the capsule is occurring.

as innocent papillomata, but the activity of the second case is decidedly suspicious. (Since this article was written this patient has been seen again in the routine 'follow-up' examination. Local recurrence has taken place, confirming our suspicions.) It will be realized how difficult it is to determine where innocency stops and malignancy begins.

## 5. PAPILLIFEROUS ADENOCARCINOMA.

In the following illustrative cases increasing degrees of activity are shown.

*Fig. 73* shows a stage more advanced than *Fig. 72*. In this patient a nodule was present in the isthmus of the gland. This was causing discomfort altogether out of proportion to the size of the nodule. The nodule was removed rather under protest, but patient and doctor were both insistent about the reality of the discomfort. Microscopical examination shows a luxuriant growth of papilliferous nature, and in many places the simple benign appearance has been lost, in that the cells covering the papillæ form multiple layers and the epithelium is growing without restraint. This had passed beyond the stage when it could be classed as a benign neoplasm.

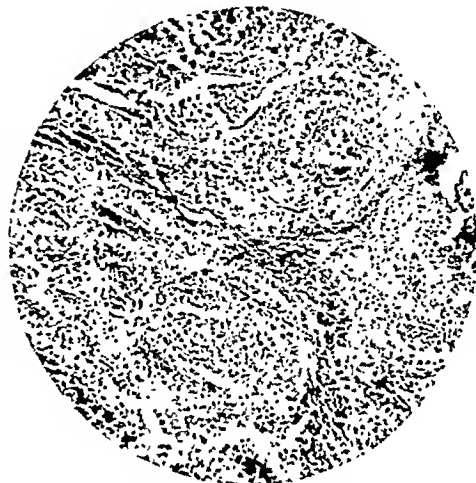


FIG. 73.—Early papilliferous adenocarcinoma.

Next in the series is a tumour in a woman of 64. In this patient growth was more rapid—it had only been noticed eight months previously—and the increase in size had been obvious. The tumour had become fixed to the adjacent structures, and neighbouring lymph-nodes were enlarged. *Fig. 74* shows papilliferous growth throughout the tumour, but sections taken from the surface of the growth where it was attached to the overlying structures show that it had penetrated the capsule and invaded muscles (*Fig. 75*).



FIG. 74.—Papilliferous growth throughout the greater part of the tumour.



FIG. 75.—From the same patient as *Fig. 74*. Growth has invaded muscle bundles overlying the tumour.

In the third patient in this series the same papilliferous change in the tumour is shown (*Fig. 76*). Malignancy was not suspected before opera-



FIG. 76.—Papilliferous change in the third patient.



FIG. 77.—From the same patient as *Fig. 76*. The growth has become more embryonic in character.

tion, and recurrence occurred. On examining the material removed at the second operation, it was found that the greater part of the mass was still papilliferous, but growth had become much more active, and areas were found where dedifferentiation or anaplasia had occurred (*Fig. 77*). The cells were invading the capsule and lymph spaces (*Fig. 78*).

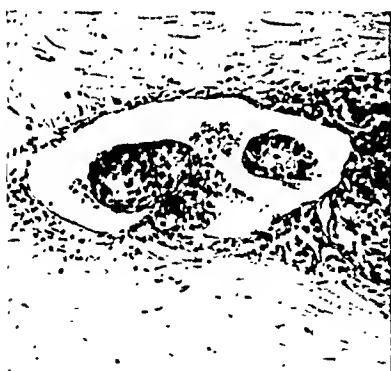


FIG. 78.—From the same patient as *Figs. 76* and *77*. Carcinoma cells in a lymph space.

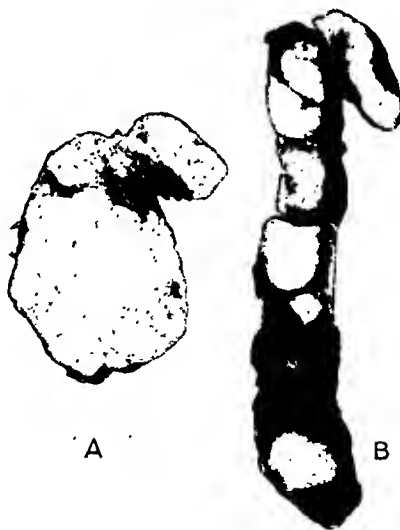


FIG. 79.—Papilliferous adenocarcinoma. A, The thyroid gland; B, Infected cervical lymph-nodes.

In the next example of this type of carcinoma, the thyroid gland and lymph-nodes throughout the neck were extensively involved. *Fig. 79* shows

the original tumour and a chain of infected cervical lymph-nodes removed at the first operation. *Fig. 80* shows the microscopical appearance of the original tumour. Section of one of the lymph-nodes shows papilliferous growth replacing the original structure with the exception of a little lymphoid tissue which remains under the capsule (*Fig. 81*).



FIG. 80.—The microscopical appearance of the tumour shown in *Fig. 79, A*.



FIG. 81.—The microscopical appearance of one of the lymph-nodes (*Fig. 79, B*).

#### Discussion of Papilliferous Adenocarcinoma.—

1. It would appear from a study of these illustrations that varying degrees of papilliferous change may be induced in thyroid epithelium.

2. In puberty goitre and in toxic goitre this is probably a response to stimulation, the type of stimulation differing in degree rather than in kind, and in both types it is probably compensatory in nature and occurs because under special stresses the function of the gland is not adequate to the demands made upon it.

3. In both puberty goitre and in toxic goitre the papilliferous condition disappears if gland function becomes normal. The cause is, to some extent, known and under control.

4. An examination of *Figs. 68–81* shows that a progressive degree of epithelial activity can be traced from the alleged simple papilloma to an invading and disseminating growth. In these conditions the cause is not known, but the histopathological appearance is, in many instances, so similar to that in which the cause is to some extent known (compare *Fig. 68* with *Figs. 70* and *74*), that we cannot help asking ourselves whether the epithelial hyperplasia forming a papilliferous neoplasm may not result from disordered physiological stimulation of a normal gland, or possibly normal stimulation directed on to a gland—or some portion of a gland—which is subesficient through maldevelopment.

5. These considerations give some reasons for the hypothesis that the epithelial response which appears to us to result in one instance in an

innocent condition may be akin to that which in another results in a malignant condition, the epithelial hyperplasia being an expression of an inherent capacity of the thyroid epithelium, and its activity being aroused by stimulation which may be normal or abnormal.

6. The work of Marine and Lenhart on brook trout, which has already been mentioned, is interesting in this respect. For many years it had been known that enlargement of the thyroid gland often occurs in brook trout when overcrowding takes place. These workers<sup>11</sup> made exhaustive studies of this condition. The goitre is conspicuous. It consists of epithelial cells of thyroid origin which spread widely but locally. The cells infiltrate and replace muscle bundles and bone in the floor of the mouth and pharynx, and may extend on to the gills. Because of the actual invasion of muscle and bone, the condition was long believed to be adenocarcinoma. In fish the thyroid gland is not encapsulated, and therefore, when hyperplasia occurs as the result of stimulation, the spread of the cells is not limited. When the cause of the hyperplasia is removed, the goitre disappears. Marine and Lenhart decide that, although local invasion occurs, this is not carcinomatous, but a hyperplasia simulating carcinoma because of the peculiar characters of the thyroid gland in fish. Nevertheless in these goitres a true tumour sometimes occurs—papillomatous in type—and this tumour does not disappear with the improvement in hygiene. The cause of this tumour in trout would seem to have something in common with that of the hyperplasia in human beings, and it will be of interest to refer to it again after consideration of lateral aberrant thyroids.

7. Lateral aberrant thyroids. A study of the behaviour of this condition (of which we have had four cases—to be described immediately) gives some suggestion that the response of thyroid epithelium—right up to malignancy—represents an interaction between stimulation and the responsive capacity of the cells, the gland efficiency in the case of lateral aberrant thyroids being limited by imperfect development. It is possible that the information gained from these structures, taken together with the experiments of Marine and Lenhart on trout—admitting that the hyperplasia in brook trout stops short of carcinoma—may help a little way towards an understanding of the evolution of papilliferous adenocarcinoma.

#### 4. LATERAL ABERRANT THYROIDS.

Many cases of this condition have now been reported. Billings and Paul<sup>12</sup> in 1925 reported a series of 35 cases published since 1857, including one of their own. Tebbutt and Woodhill<sup>13</sup> in 1927 reported 5 cases and discussed the literature, together with the clinical condition and pathology of their own cases. In 1928 L. van den Wildenberg<sup>14</sup> reported a case of his own together with 28 cases collected from the literature. In the same year the workers in the Lahey Clinic<sup>15</sup> had collected records of 45 cases, including 4 of their own. In the published cases there have been one or more tumours, usually in the posterior triangle, sometimes situated just above the clavicle, sometimes high in the neck.

We have had four cases of this condition. In each, multiple tumours

have been present, and these have been quite distinct from the thyroid gland, although in all our cases the thyroid gland itself has been enlarged.

**Origin of Lateral Aberrant Thyroids.**—Whence came these nodules embryologically, and what are they pathologically? At first, because of the lymphoid tissue under the capsule, the colloid vesicles, and the papilliferous growth, it was thought that they were deposits of thyroid carcinoma in lymph glands; but it would seem that these masses are aberrant thyroid tissue, or derive from the pharyngeal entoderm. Morphologically these cystic papilliferous aberrant thyroids are very similar to papilliferous adenocarcinoma occurring in the thyroid.

Writers in Europe and America refer to lateral aberrant thyroids as probably deriving from the ultimo-branchial body—the remnant of the fifth pharyngeal pouch. This belief is not altogether based on demonstrable facts. Langhans had this opinion, and thought that papilliferous adenocarcinoma occurring in the thyroid gland had the same origin. L. van den Wildenberg<sup>14</sup> and the workers in the Lahey Clinic<sup>15</sup> regard this as a strong probability. Wegelin discusses the question at length in the *Handbook on Special Pathology* by Henke and Lubarsch, and in another article in the *Cancer Review*<sup>7</sup> he states: “It appears in most cases to be derived from isolated branchiogenetic rests, especially when it occupies the lateral cervical region.” Kingsbury<sup>16</sup> and Badertscher<sup>17</sup> have shown that the ultimo-branchial body becomes fused with the developing thyroid gland in the pig. Grosser<sup>18</sup> (Keibel and Mall) considers it probable that in the migration of cells from the posterior portion of the pharynx to the developing thyroid complete fusion has failed to take place and rests remain. Even though fusion takes place, development may remain imperfect. Williamson and Pearse<sup>9</sup> state that from their view of the development of the thyroid, they expect to find in certain imperfectly developed human glands states corresponding to the more primitive types found in some lower animals. In these conditions, they would expect abnormality of function, and a consequent tendency to malignancy, for they look for one of the predisposing causes to all neoplasia in anatomical and functional abnormalities. The association of lymphoid tissue containing Hassall’s corpuscles with this type of thyroid growth is also explained by them on an anatomical basis, for these authors find evidence in the thyroid apparatus of two distinct and separate lymph systems. One of these—the intrinsic thyrothymic lymph system—links the thyroid directly with the thymus, so that the thyroid and the thymus (including the nodes of the thymus scattered in the neck) come to be regarded by them as functionally one organ. Following this theory, they interpret this type of carcinoma as a growth confined, until its later stages, within the lymphatics of the *organ of its origin*, and they believe that this accounts for its anomalous nature, its slow growth, absence of distant metastases, and widespread growth in the mediastinum.

The various theories have been discussed by Professor Woollard,<sup>19</sup> and he has shown that this aberrant tissue need not necessarily derive from the ultimo-branchial body; the potency of the pharyngeal entoderm is such, and the developmental processes are so complicated in that small area, that colloid-producing epithelium attached to or surrounding lymphoid tissue

which may contain Hassall's corpuscles, may be displaced by developing nerves, muscles, or vessels, and lodge in the lateral aspects of the neck.

For my purpose it is not necessary to decide on the actual origin of the entodermal rudiments. The solution of this must remain in the hands of the embryologists, but it is an important question whether these laterally placed rudiments are imperfectly developed, whether tissue of similar origin is incorporated in the thyroid gland, and whether the imperfect development of this tissue leaves it susceptible to malignant degeneration. We are therefore keenly interested in the pathological histology, for it would seem that these misplaced bits of tissue, whether in the lateral triangle of the neck or in the thyroid gland itself, foredoom the host to carcinoma from before the day of birth.



FIG. 82.—Case 1. Lateral aberrant thyroids. The tumour situated in the left posterior triangle where the neck joins the shoulder was very conspicuous.

I had the patient photographed before part of the gland. The masses were cystic and papilliferous, and the gland was of the same nature. Fig. 82 shows the nodules in the posterior triangle of the neck on each side.

Case 2.—The patient was 46 years old when I saw her, and she gave a history of at least eight years, so that the condition dated from her early thirties. Masses were present high in the neck behind the angle of the jaw, as well as in the posterior triangles. Fig. 83 shows a section from a nodule removed. Paralysis of a recurrent laryngeal nerve was present when this patient was first seen by me.

Case 3.—The patient (Fig. 84) was 24 when admitted to St. Bartholomew's Hospital, and the condition had been present for seven years, so that it had begun before the age of 17. Several of these nodules were removed. A complete section of one of them is drawn in Fig. 85. Lymphoid tissue is present under the capsule. The histology is shown in Fig. 86.

#### Clinical History and Pathological Histology of our Four Patients.—

Case 1.—The patient was a young man upon whom I operated in Australia some years before the war. It was very early in my surgical life, and I fear I was not then as interested in keeping detailed records as I should have been. excising the lateral masses and a large cystic and papilliferous, and the gland was



FIG. 83.—Case 2. Section from a lateral aberrant thyroid.



FIG. 84.—Case 3. Lateral aberrant thyroid. Thyroid gland also enlarged.

FIG. 85.—Case 3. Lateral aberrant nodule. In the stained section lymphoid tissue is clearly seen under the capsule. ( $\times 2$ )

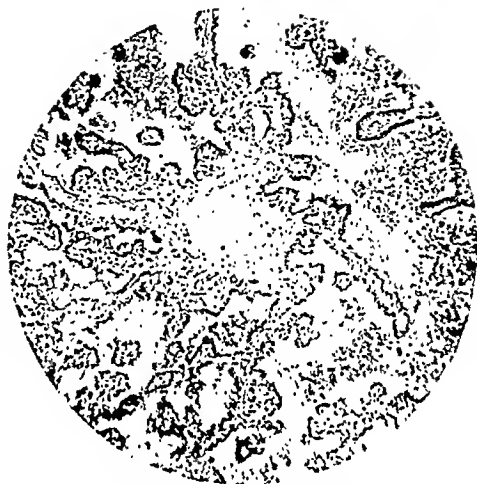


FIG. 86.—Case 3. Microphotograph of section taken from the centre of the nodule shown in Fig. 85.



FIG. 87.—Case 4. Lateral aberrant thyroids. The thyroid gland was also enlarged.

Case 4.—The patient (Fig. 87) was 16 when admitted to St. Bartholomew's Hospital, and the condition was known to be present at the age of 12.

In each of these four the condition would appear to be identical. There were nodules of tissue situated in the posterior and anterior triangles of the neck, encapsulated, having no connection, but associated with an enlargement of the thyroid gland. Microscopically the nodules consist of papilliferous growth into cystic spaces, some of which contain colloid, and immediately under the capsule the remains of lymphoid tissue. These tumours are usually classed as papillary cystadenoma with a tendency to undergo malignant transformation.



Taking our four patients: The first was operated upon about eighteen years ago in Australia. I have been unable to trace him since the beginning of the war. The second patient had, when I saw her, involvement of a

recurrent laryngeal nerve with paralysis of a vocal cord, implying that the growth was infiltrating. She ultimately died of intestinal obstruction following malignant disease in the pelvis. The third we were able to keep in touch with for three years. She married and had two children, but she regarded our interest in her as far too pathological, and all our efforts to find her subsequently have proved unavailing. The fourth patient was only 16, and the tumours had already been present for at least four years. One nodule was removed for diagnosis. The patient was friendly and interested, and we felt that we were going to be able to keep her under observation indefinitely, but within nine months of the time we first saw her, symptoms of pressure on the trachea began. These increased so rapidly, and were so urgent, that operation became necessary. Five nodules were removed (*Figs. 88, 89*)—making six with the one previously excised—and also the greater part of the thyroid.

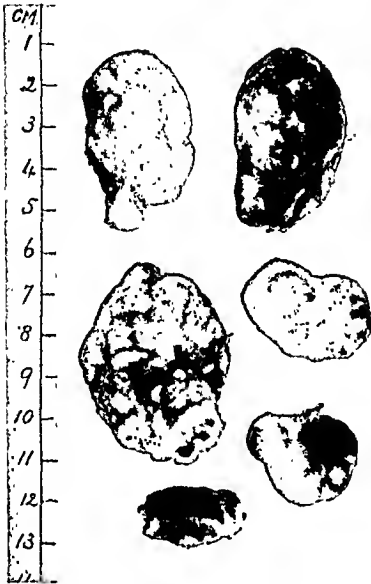


FIG. 88.—Case 4. Lateral aberrant thyroids (with the exception of the largest nodule, which is part of the thyroid gland).

Relief followed, but pressure rapidly recurred. Deep X-ray therapy failed to give relief, and the patient died.

**The Bearing of Lateral Aberrant Thyroids on the Question of Adenopapilliferous Carcinoma.**—The embryology is not settled, but it is believed that these bodies originate from the pharyngeal entoderm, and that they have failed to join the median thyroid gland, and therefore to develop perfectly.

All writers agree that cystic papilliferous change takes place in lateral aberrant thyroids, and that there is a strong tendency for this to become malignant.

In 45 cases<sup>12, 13, 14</sup> collected from the literature, and including our own, the condition became obvious in twenty-five before the age of 30, and in fifteen before the age of 20 years.



FIG. 89.—Case 4. Histological picture of one of the nodules shown in *Fig. 88*.

In the majority of these the change

was advanced when the pathological examination was made. The average age at which these tumours develop is far earlier than is usual with epithelial tumours of any other organ, but it does accord with the age at which non-malignant enlargements of the thyroid gland occur—the enlargements which we regard as compensatory or resulting from some kind of stimulation.

Papilliferous change is almost universal in lateral aberrant thyroids. It was present in our four cases, and in practically all the published cases at the time the patient was first seen. (Mr. Cecil Joll has very kindly shown me a section from a tumour removed by the late Mr. Charles Ryall in which papilliferous change had not occurred.)

The early age (adolescence in some) of the patients, and the very high frequency of the papilliferous change, almost compel the question whether the gland tissue of lateral aberrant thyroids does not undergo papilliferous hyperplasia under the stimulation of the ordinary physiological demands.

In the body there is always interaction—and usually balance—between stimulation and capacity for response, but in the thyroid gland we frequently see this equilibrium disturbed, on the one side through variation in the intensity of the stimulation, and on the other through variation in the capacity of the organ to respond. If the physiological demands of the body could be kept at a lower level, the lateral aberrant thyroids might remain as imperfectly migrated 'rests'. If the gland tissue of which they consist were more completely developed, it might still function without tumour formation. In this respect it is interesting to revert to the tumours occurring in brook trout, where a condition in many ways so nearly approaching carcinoma can be induced in this very labile organ by the appropriate stimulation.

### MALIGNANT ADENOMA.

'Malignant adenoma' is not a perfect term, but in the present state of knowledge it would appear to be the most satisfactory. It is meant to convey that malignant transformation has occurred in an adenoma, and that in some respects the tumour maintains the morphological characters of an adenoma; and by the term 'adenoma' is meant the benign neoplasm usually called in this country 'foetal adenoma'. Aschoff<sup>5</sup> has shown that adenomata arise from proliferation of adult cells, not from foetal rests as was formerly believed. The capsule is formed by condensation of the stroma, and by compression of neighbouring tissue. The term 'foetal' is therefore not correct if it is meant to indicate the origin of the tumour. It may be used to indicate that the tissue is more embryonic in appearance than normal gland.

This type of carcinoma constitutes a high percentage of the total numbers. If an average is taken from large clinics it includes 85 to 90 per cent of the total.

Benign adenomata differ from one another in histological appearance, though they are always clearly distinguished from involution nodules. Some are composed of anastomosing columns of cells (*Fig. 90*). In others differentiation has occurred, and the tumour consists of follicles. Many of these are immature, containing no colloid, and scarcely a lumen, while other follicles are more mature and contain colloid (*Fig. 91*).

When malignant transformation has occurred, these ancestral characteristics are maintained to a considerable extent, although ultimately—early in some, but very late in others—differentiation is lost and the cells grow



FIG. 90.—Benign adenoma showing anastomosing columns of cells forming miniature tubules.

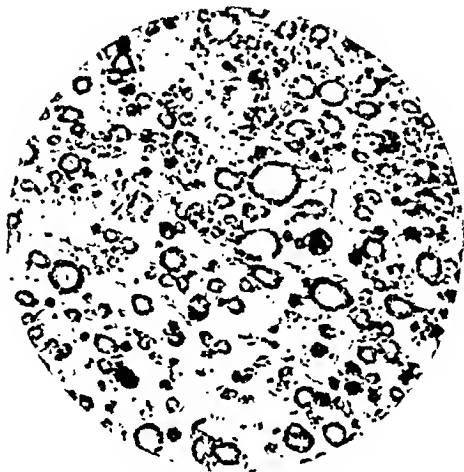


FIG. 91.—Benign adenoma. Some vesicles contain colloid, some have no lumen, others are quite immature.

in disorderly fashion. The histological appearance of malignant transformation that has taken place in the first type of adenoma is shown in *Fig. 92*. This is from a deposit under the skin, but superficial to the sternomastoid muscle, in the scar of the operation for the removal of the adenoma. In this the anastomosing columns are still clearly seen, although growth is more active. It may be asked: Is this really malignant? *Fig. 99* shows deposits in the lungs in this patient.

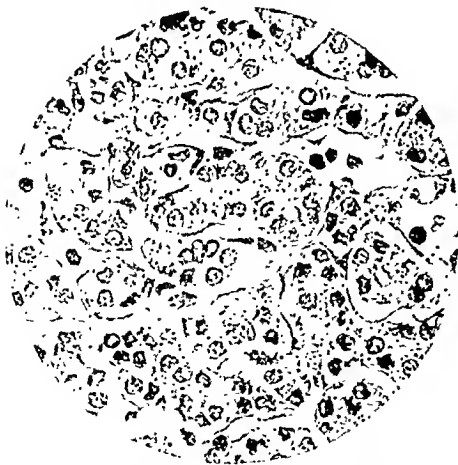


FIG. 92.—Malignant adenoma arising from a tumour of the type shown in *Fig. 90*.

An example of the second type—that is, the retention of the follicular structure of the parent adenoma after malignant transformation had taken place—is illustrated by *Fig. 94*. Again it may be asked: What are the criteria of malignancy, for the follicles in some parts are as highly differentiated as in some non-malignant adenomas? In this patient the growth has penetrated veins and filled the large vessels on the surface of the gland; not only

so, but it has transgressed the capsule and invaded the tissues of the neck. A blood-vessel completely filled with tumour growth is shown in *Fig. 93*. The growth filling this vessel retains its follicular structure (*Fig. 95*).

These morphological differences, together with some biological characteristics, have led Continental observers to magnify subdivisions of malignant adenoma into definite types of carcinoma under the names of 'proliferating adenoma' and 'metastasizing adenoma'. This nomenclature appears to be unnecessarily complicated and mystifying, for in malignant adenoma every cellular type is found between a high degree of differentiation on the one hand and complete de-differentiation or anaplasia on the other. To a great extent the degree of differentiation is in inverse ratio to the grade of malignancy. When differentiation is lost, the highly cellular growth gives the appearance conventionally called 'medullary' (*Fig. 96*), and many authors have given this name to a type of carcinoma. This also is unnecessary. It does indicate a grade of anaplasia, but not a different class of carcinoma.



FIG. 93.—Section of a vessel from the surface of the tumour illustrated in *Fig. 94*. ( $\times 3$ .)

Malignant adenoma not only differs morphologically from papilliferous adenocarcinoma, but its method of dissemination is different. We have seen that papilliferous adenocarcinoma disseminates primarily to adjacent lymph-nodes. Malignant adenoma does not disseminate to lymph-nodes while

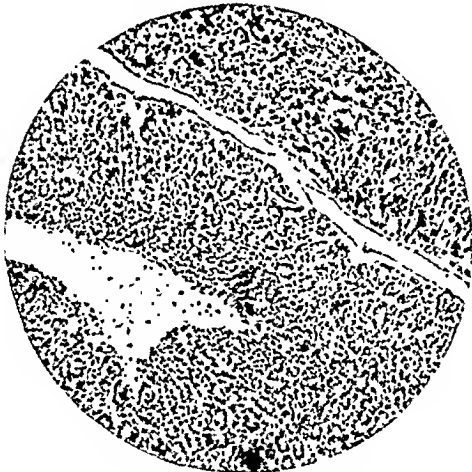


FIG. 94.—Malignant adenoma retaining follicular structure.



FIG. 95.—Microphotograph of growth filling the vessel shown in *Fig. 93*.

contained within its capsule, but even at this stage it invades blood-vessels, and therefore distant metastases may occur while the primary growth is small. This has given rise to the theory, so long believed, that metastases could originate from a normal thyroid gland, or from small benign adenomata. Invasion of blood-vessels is almost certainly responsible for the deposits seen in *Fig. 99*. As the percentage of cases of carcinoma of the thyroid gland originating in benign adenomata is so high, it is important to try to

decide when the change is taking place. *Fig. 97* is a microphotograph of a section of an adenoma removed from a patient in whom malignancy was unsuspected, the nodule having been present eight years and enlarging

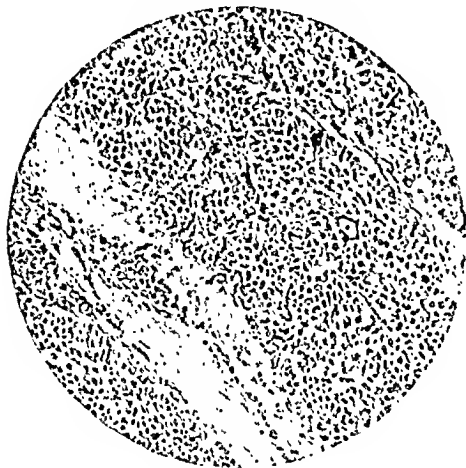


FIG. 96.—Malignant adenoma in which structure has been lost.

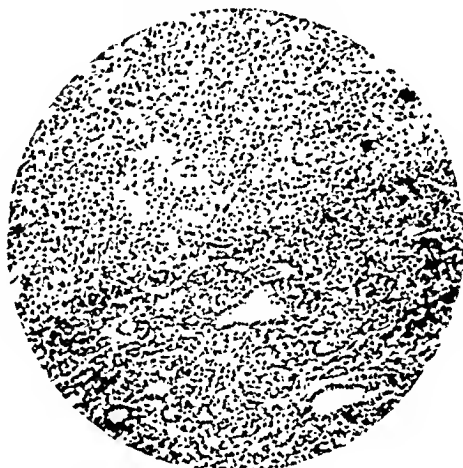


FIG. 97.—Microphotograph of section of an adenoma removed eighteen years ago. It was then considered to be suspicious of malignancy.

slowly. The section is not very clear, for it was cut and stained eighteen years ago and with no idea of reproduction. The cells appeared to be unduly active for an innocent tumour, but could not be diagnosed as definitely malignant. The tumour was classed as suspicious, and the patient kept on a follow-up list. Five years later a nodule appeared under the skin in the scar over



FIG. 98.—Nodule removed from the extreme end of the incision, over the sternomastoid muscle. The skin is seen superficial to it. This is from the same patient as *Figs. 92, 97, 99*.

the sternomastoid muscle. Ten years from the time of removal of the original tumour a similar nodule appeared. *Fig. 98* shows the whole nodule with the skin stretched over it, and *Fig. 92* the histological appearance. Four years later—fourteen years from the original operation—severe hæmoptysis led to an X-ray examination of the lungs being made. Deposits were found to be present in both



FIG. 99.—Deposits in both lungs. From the same patient as *Figs. 92, 97, 98*.

lungs (*Fig. 99*). Therefore unusual activity of epithelial cells should put us on guard and make us watch for biological evidence.

The varied changes that may occur in one adenomatous nodule are well illustrated in *Figs. 100-103*. They are from a patient in whom a tumour had

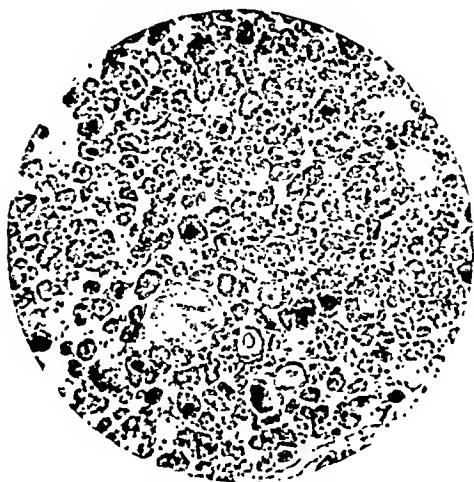


FIG. 100.—This section by itself is consistent with the tumour being a benign adenoma.

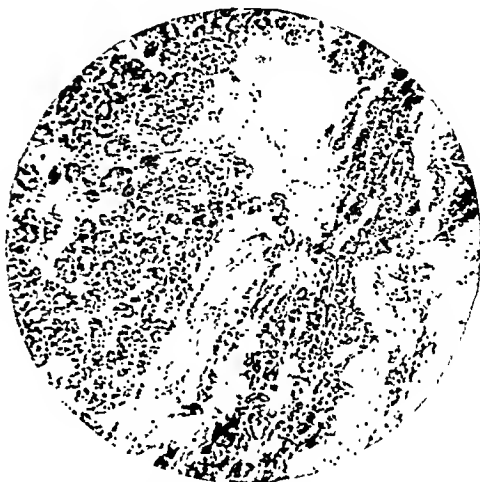


FIG. 101.—The epithelium, while retaining its follicular arrangement, is invading and penetrating through the capsule. From the same case as *Figs. 100, 102, 103*.

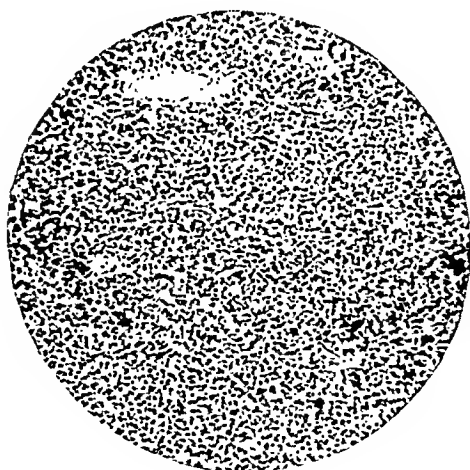


FIG. 102.—This is still suggestive of vesicle formation, but structure is practically lost and the epithelium more embryonic. From the same case as *Figs. 100, 101, 103*.



FIG. 103.—A small area of papilliferous growth occurring in the same tumour as *Figs. 100-102*.

been present for eighteen years and shortness of breath for one year. The enlargement had been present so long that she had become accustomed to it. The first (*Fig. 100*) shows a typical 'foetal adenoma'. Many of the vesicles are well formed and contain colloid. Others are immature and without lumen. *Fig. 101*, from the same patient, shows invasion of the capsule even while

the cells are retaining their follicular arrangement. It may be objected that the capsule is growing in and strangling the tumour. I think that this is not so, for the nuclei all stain well; also the following sections from other parts of the tumour give confirmatory evidence.

*Fig. 102*, from another part of the same tumour, shows the arrangement of the epithelium becoming much more active and embryonic in character.

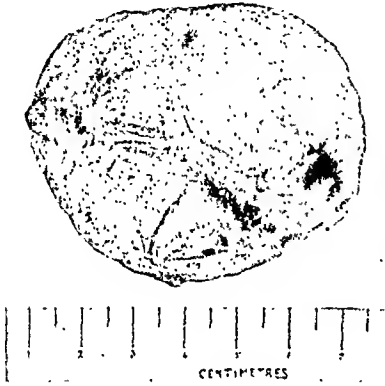


FIG. 104.—Adenomatous nodule.

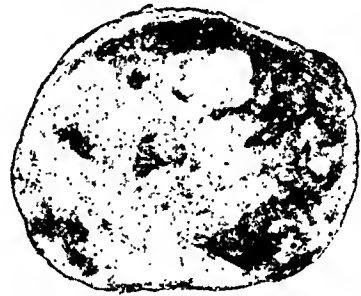


FIG. 105.—Cross-section of the nodule shown in *Fig. 104*.

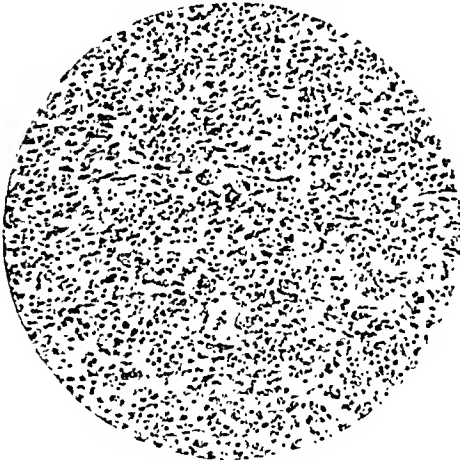


FIG. 106.—Microphotograph of one area of the nodule in *Fig. 104*, showing malignant change.



FIG. 107.—Microphotograph of another area of the nodule in *Fig. 104*, showing malignant change with different characters.

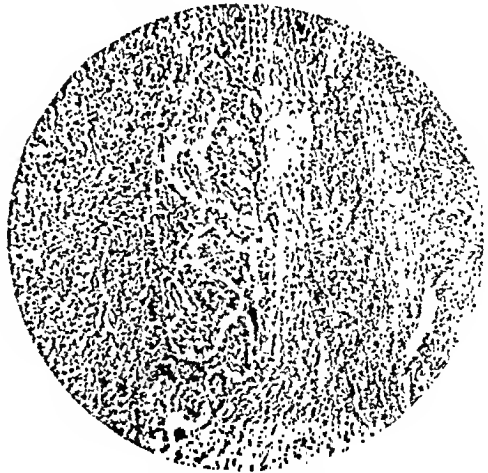
Such vesicles as exist are immature, contain no colloid, and many contain no lumen. The epithelium is breaking away from vesicle formation altogether. In still another part papilliferous growth has occurred (*Fig. 103*). Add to all this epithelial activity the fact that this tumour—not large in itself—could not be dissected off, but had to be cut away from the upper rings of the trachea and the thyroid cartilage, and I think there can be no doubt that malignant change had taken place.

The change within the capsule may be very extensive before clinical evidence of invasion becomes obvious, or any suspicion of malignancy is aroused. The nodule shown in *Fig. 104* had been known to be present in the thyroid gland of the wife of a medical man for six years. Its lower margin just dipped beneath the left sterno-clavicular articulation. It could be freely moved, but it had begun to cause pressure symptoms, and it was conspicuous. The patient disliked the look of it, and her husband realized that the pressure symptoms were interfering with her comfort. It was cleanly removed. On cutting across the specimen, suspicion was aroused (*Fig. 105*), and on microscopical examination very active malignant change was revealed (*Figs. 106, 107*).

**Advanced Cases.**—I have not considered advanced cases of malignant adenoma. That would serve no useful purpose at this part of the discussion. In the late stages of the disease specific glandular structure will generally have disappeared, and the histopathological appearance will be that shown in *Figs. 96, 111, and 113*. When discussing classification it was stated that the second and third types of carcinoma tend to overlap. This is shown in *Fig. 103*, where in a characteristic malignant adenoma one papilliferous area was found. Thyroid epithelium seems never to lose this capacity entirely. In the case shown in *Figs. 108, 109* the growth consisted of sheets of cells which in many places were suggestive of sarcoma; yet in one microscopie field a tiny area of papilliferous formation is seen. This rarely prevents each type from being readily elassed. Again, in the later stages of papilliferous adenocarcinoma an area will sometimes be found where the epithelium is growing in broad sheets (*see Fig. 77*). The classification must not be made too rigid.



**FIG. 108.**—Carcinoma of thyroid, malignant adenoma type.



**FIG. 109.**—A small papilliferous area in the tumour shown in *Fig. 108*.



**Discussion on the Origin and Pathology of Malignant Adenoma.**—The origin and evolution of adenomata has been described by Aschoff.<sup>5</sup> From his detailed description it cannot be doubted that they occur as the result of some form of stimulation or irritation. In a small percentage of these, epithelial activity crosses the line dividing innocency from malignancy. Although this percentage is small, it constitutes by far the biggest class of carcinomas of the thyroid. How long the epithelial activity has been taking place before any evidence of it becomes obvious clinically it is impossible to say, and it will have been realized that the interpretation of the histopathology presents great difficulty. This is because the epithelial appearance of different benign adenomas, or different parts of the same adenoma, may vary so widely, and because malignant change may take place with so little alteration in the characters and arrangement of the epithelium. Graham and Wegelin have stated that neither the character of the cells, nor the mitoses, nor the structure of the vesicles admits of any definite conclusion as regards non-malignancy or malignancy, and we heartily agree with the latter when he states that “from a pathological standpoint the dividing line between non-malignant and malignant tumours can probably nowhere be drawn with greater difficulty than just here.”<sup>20</sup> Graham<sup>21</sup> considers vascular invasion the chief test of malignant change in an adenoma. Sometimes (*see Figs. 93, 95*) the invaded vessels are obvious on the surface of the growth; sometimes they are only found after patient search under the microscope. In malignant adenoma neighbouring glands are not infected while the growth is contained within the capsule—in this respect it differs from scirrhous and papilliferous adenocarcinoma—but the invasion of veins may occur very early and while the growth is still encapsulated. As a consequence of this, distant metastases may occur—to lungs or bones—while the parent growth is small. This invasion of veins while the primary growth remains small, together with the fact that secondary deposits retain the characters of the primary tumour, is the explanation of the statement that metastases may arise from benign tumours. We have not had an example of an *inconspicuous* tumour giving rise to lung metastases while no invasion of lymph-nodes in the neck was ever found (*see Fig. 99*).

In spite of the difficulty sometimes found in interpreting the histopathology, I believe that the sections presented have shown changes that it is impossible to ignore. The search must be thorough, and sections taken from several places. Remembering the various types of epithelial arrangement that may occur in non-malignant tumours, all the evidence must be considered, including epithelial activity, mitosis, the beginning of alteration in structure, sometimes invasion of capsule or vessels even when specific glandular structure is retained. Seeing that the question is not a purely academic one, since we are dealing with human beings, all clinical evidence must be taken into account.

### CLINICAL COURSE OF CARCINOMA OF THE THYROID GLAND.

1. **Scirrhous or Carcinoma Simplex.**—This type does not differ in its habits from the same type of carcinoma occurring elsewhere.

2. **Papilliferous Adenocarcinoma.**—The evolution of this type is without doubt slow in the majority of cases, but it is inevitably progressive. Sir James Berry<sup>22</sup> wrote that these tumours grow slowly and are more amenable to treatment by removal, even though they may have attained large size. He cites Mr. Barker's patient<sup>23</sup> who lived eighteen years after the first appearance of the tumour. This patient underwent numerous operations, but the growth caused his death in the end. The fact that growth is slow does not make these tumours any the less malignant. Among the cases illustrated it will be remembered that even in a papilloma regarded as benign the epithelial activity was suspicious, and that in successive cases invasion of adjacent tissues and deposits in lymph-nodes occurred.

3. **Malignant Adenoma.**—In this condition it has generally been assumed that growth is more rapid, but even in this type it varies within very wide limits. One patient died within twelve weeks from the time she first recognized that her adenoma had begun to cause discomfort, while another is alive eighteen years after removal of a tumour diagnosed as malignant, and proved by the subsequent history of the patient to have been so (*see Figs.*



FIG. 110.—Malignant adenoma, intrathoracic. Skiagram taken ten years after the removal of the original tumour from the neck. This tumour was removed after splitting the sternum. Recurrence in the chest took place quickly.

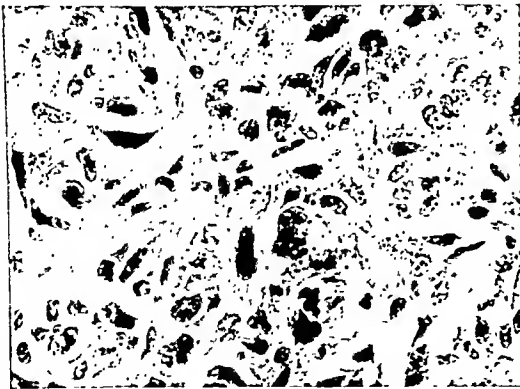


FIG. 111.—Microphotograph showing histopathology of tumour shown in Fig. 110.

92, 98, 99). The following case lies between these two extremes:

A mass had been removed from the neck on account of extreme dyspnoea ten years previously in another hospital, and it was pronounced malignant by the hospital pathologist; that the diagnosis was correct was proved by the later history. The mass removed by me at this late stage was undoubtedly malignant (*Figs. 110, 111*). Recurrence took place, and the patient subsequently died, but ten years had elapsed before the tumour began to grow rapidly.

In the patient from whom the sections shown in *Figs. 93–95* were taken the tumour had been present for fifteen years. Some increase in size one year previously had not disturbed him, but he began to be rather irritated by the constant necessity for clearing his throat of phlegm and the difficulty in doing so. Large surface vessels were invaded with growth. The tumour

from which *Figs.* 100–103 were taken had been present for eighteen years. It is not possible to know when the change had begun in the last two cases, but in the two preceding these there is microscopical evidence of epithelial activity extending over eighteen and ten years respectively. I therefore feel sure that in many cases malignant adenoma is comparatively slow in its evolution. In its earliest stages it is confined within the capsule of the adenoma, and how long it is from the time the first change begins in the epithelium until the capsule is penetrated, it is impossible to say. Certainly it will have begun before increase in size of the nodule is obvious. It precedes—for it causes—the pressure symptoms and fixation. The first suspicion of change will be aroused by clinical signs, and then all the evidence should be considered. If the tumour is removed, the most careful examination must be made, and for this blocks of tissue must be chosen from several parts of the tumour. Often enough the diagnosis is dated from a period subsequent to penetration of the capsule, when the induration, the nodular appearance, and the pressure symptoms leave no doubt of the condition.

### PROGNOSIS.

Malignant disease of any kind offers no cheerful outlook, and malignant disease of the thyroid gland has been regarded rather hopelessly. Looking through some recent text-books I find that operation is only advised in the early stages—that is, when the outline is still clear, mobility present, and enlarged glands or secondary deposits are absent; also the mortality of operation is placed as high as 25 to 30 per cent; that most cases are inoperable when first seen, and that radium and X-ray treatment have little effect on the growth; that the growth should still be within the capsule of the gland and must not be adherent to the trachea; and that (when commencing in an adenoma), if local malignancy is clearly shown by signs, no operation should be done, but if there is doubt, operation should be performed on the chance of a cure.

It is true that in scirrhus any lasting benefit is unlikely to be obtained; but scirrhus is rare, and even in this type it is a hopeful sign that the tumour from which *Fig.* 66 was taken was temporarily reduced in size by at least half by the use of radium needles. In the two other types—and they constitute the vast majority—the outlook should be less gloomy for the following reasons:—

1. Diagnosis is made—or suspicion aroused—much earlier than was the case formerly.

2. The rate of growth in papilliferous adenocarcinoma is slow. In many cases of malignant adenoma it is also slow, and is for a long time retained within the capsule. (It is to be borne in mind that dissemination by the blood-stream may occur before invasion of the capsule.)

3. Experience is showing that malignant epithelial cells of thyroid origin are radio-sensitive. I say this after making every allowance for error of judgement on account of the slow rate of growth in many cases.

To inform a patient with a hard nodule that it is unimportant and should be ignored is not right. It is not our function to operate upon every

abnormal thyroid, but when our advice is sought regarding a lump, it is our function to weigh all the evidence and to advise the patient wisely.

Again, even in late cases, it is possible to plan and carry out an operation which may give great relief in itself and may make further relief possible by X-ray treatment.

In our series no patient has died at operation, although in some the condition has been advanced, causing the patients great distress. In several patients the sternum has been split to obtain access to a deep thoracic mass (*Figs. 110, 112*), and in others a large fixed mass has surrounded the neck, involving the deep structures and invading the upper inlet of the thorax to an extent that made breathing extremely difficult.



FIG. 112.—Malignant adenoma invading the superior mediastinum. The greater part of this was removed after splitting the sternum.

### RADIO-SENSITIVITY.

#### X RAYS.

The first patient in whom the value of X-ray treatment was brought home to me was seen in 1922. The patient had a large tumour encircling the front of the neck, with extension into the thorax. The extent of the intrathoracic growth is shown in *Fig. 112*, and with the massive growth in the neck it will be realized that respiration was greatly hampered. Because of the fixation of the growth, access to the intrathoracic portion could only

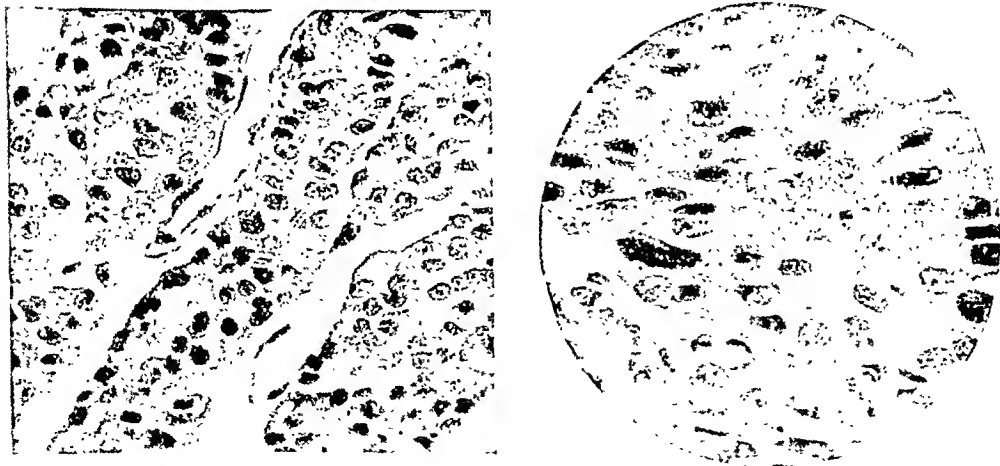


FIG. 113.—Microphotographs from two portions of the tumour removed from the patient whose skiagram is shown in *Fig. 112*.

be obtained by splitting the sternum. Growth was cut across both in the thorax and in the deeper planes of the neck. The appearances of the tumour are shown in *Fig. 113*. It was of malignant adenoma type. Because of the growth known to be left, X-ray treatment was given, though this was not deep X-ray therapy in the modern meaning of the term. A subsequent skiagram (*Fig. 114*) shows the contrast in the upper thorax. The patient resumed his work and carried it on for two and a half years. Then secondary deposits in the skull revealed themselves, and cachexia and death followed. The growth was a malignant adenoma of medullary type.

In another patient, seen in 1926, the growth was a malignant adenoma. It had transgressed the capsule, and was firmly fixed. The histopathology is shown in *Fig. 96*. Dyspnoea and dysphagia were severe. She could only

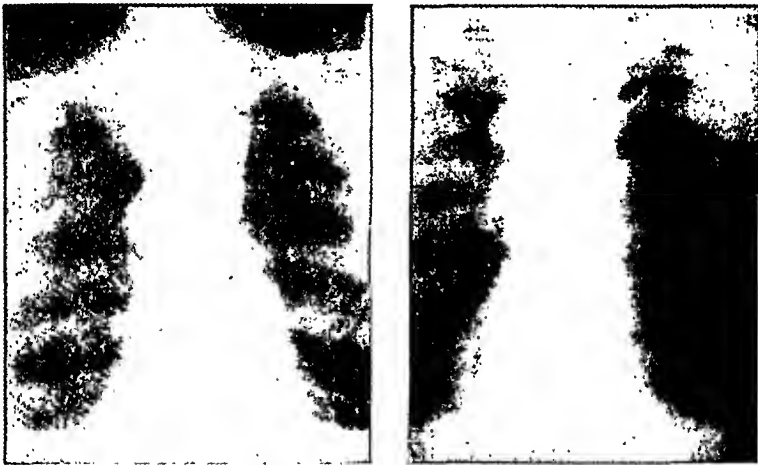


FIG. 114.—The appearances of the superior mediastinum before and after excision of the tumour shown in *Fig. 112*, and subsequent X-ray treatment.

swallow soft foods and liquids. Local recurrence followed operation. Deep X-ray therapy appeared to bring about disappearance of the recurrence. This patient died four years later from cerebral abscess. A small area of growth had eroded the common carotid artery.

Another patient, operated upon in 1927 for malignant adenoma (*Fig. 115*), is still alive and apparently well, except for paralysis of one vocal cord. This was due to damage to the nerve at operation, for the growth was extensive and much dissection was necessary.

These cases were of malignant adenoma type. The two following are examples of the result obtained in the papilliferous adenocarcinoma type.

*Figs. 79–81* are from a patient with carcinoma in the thyroid gland and deposits in the lymph-nodes of the neck, seen and operated upon in 1924. The late Mr. Barker and Sir James Berry in this country have kept patients alive for many years by multiple operations, but the disease ultimately resulted in death. More recently cases have been reported in which, when the disease has been operated upon early the patients are still alive.<sup>3</sup>

In the case under discussion, diagnosis was not made early. The patient underwent two operations, but lymph-nodes continued to enlarge. X-ray treatment was commenced at St. Bartholomew's Hospital, and the nodes disappeared. Now, seven years subsequently, this patient is robust, there is no sign of growth, and for several years she has been at full work.

*Fig. 116* is a microphotograph from a patient who was seen and operated upon in 1924. An irregularly shaped tumour of the thyroid was present. It had penetrated the capsule and become fixed to the overlying structures. Dyspnoea compelled her to seek treatment. The greater part of the tumour was removed, but its removal was known to be incomplete. The illustration shows groups of carcinoma cells lying between muscle bundles. X-ray treatment was given. Now—seven years later—she appears to be perfectly well.



FIG. 115.—Malignant adenoma. Excision of tumour and subsequent X-ray treatment.



FIG. 116.—Carcinoma cells invading the overlying muscles. This patient is well seven years after treatment was commenced.

In 14 cases X-ray treatment has been of decided benefit. Of these, 10 are still alive: 2 seven years after commencement of treatment, 2 five and a half years, 3 three years, 1 two years, and 2 others almost one year after commencement of treatment. These latter are too recent to be used as evidence.

In judging the result of X-ray treatment, the slow rate of growth in many cases must be remembered, but it can scarcely be doubted that in some of them a stage had been reached that was becoming incompatible with life. With reference to the slow rate of growth, something should be said of the patient mentioned earlier with the long history culminating in deposits in both lungs (*see Figs. 92, 97-99*) and severe hæmoptysis. No X-ray treatment had been given in the earlier years of this patient's history. The original tumour was apparently completely removed within its capsule, but—the subsequent history shows—not before cells had reached the lungs through blood-channels. Fourteen years later these had developed into multiple tumours causing severe and repeated hæmoptysis. Deep X-ray

therapy was begun at once. The hæmoptysis ceased, the patient has returned to Australia, and there have been no attacks of hæmoptysis since—that is, for three and a half years. It is possible, but scarcely likely, that when these deposits in the lungs had reached the stage of causing hæmorrhages, the hæmorrhages would have ceased spontaneously. There are ten of these patients not only alive but apparently perfectly well. Instead of needing multiple operations, the signs of the disease have disappeared, and no recurrence has taken place.

These results are not always obtained. It might be expected that penetration by X rays would be such that cases would respond whether operation had been performed or not. Our experience has been that where a large tumour has been present and none of it removed by operation, no improvement has followed X-ray treatment. In three cases no improvement has followed even after removal of the bulk of the tumour.

#### RADIUM.

Only two cases have seemed to be suitable for radium treatment. In one needles were implanted after the removal of a tumour which microscopical examination proved to be a papilliferous adenocarcinoma, and which was imperfectly removed. In the other (see *Fig. 66*) removal proved impossible because of dense fixation of a scirrhus carcinoma. No improvement followed X-ray treatment. Radium needles were then implanted (*Fig. 117*). Following this the tumour diminished in size by at least half. It subsequently increased and the patient died. The reduction in size was striking, and it shows that the epithelium in scirrhus carcinoma is radio-sensitive.



FIG. 117.—Radium needles implanted in carcinoma of the thyroid (scirrhus). This is the patient from whom *Fig. 66* was taken.

The use of radium by the insertion of needles presents difficulties. It is impossible to tell how deeply the growth has extended in the neck or into the thoracic inlet. It is likely to involve structures which it would be dangerous to puncture with needles.

In our experience the best treatment at the present time is to remove all that can be safely removed, and then have X-ray treatment given. The results even in advanced cases have often been surprisingly good.

#### DISCUSSION.

Leaving scirrhus out of this survey, for nothing has been observed which adds to our knowledge of it, we have evidence of epithelial hyperplasia

occurring in papilliferous adenocarcinoma, in lateral aberrant thyroids, and in malignant adenoma. The first two of these have so many points in common that they would seem to be practically similar conditions. Is malignant adenoma essentially different? We know that the hyperplasia which thyroid epithelium undergoes in its attempt to cope with demands made upon it, or as a response to stimulation, may be either papilliferous or follicular in type. Then we remember Aschoff's work where he points out that benign adenomata originate as tiny areas of epithelial proliferation evidently as a result of irritation or stimulation. The proliferation continues with gradual increase in size until the encapsulated nodules which we recognize clinically are produced. The majority of these remain benign, and many degenerate. In the few in which epithelial activity continues and invasion of vessels of the capsule occurs, we should like to believe that this is the result of continuance of irritation or stimulation. It does not seem unreasonable to suppose that the same factors are at work and analogous effects brought about in malignant adenoma and in papilliferous adenocarcinoma.

The investigation of our material has compelled me to place our cases in a sort of linear series by which we pass from degrees of hyperplasia which may resolve by appropriate treatment, to benign neoplasms, and finally to invading and disseminating growths. The gradation between each member of the series which along one line leads to papilliferous adenocarcinoma, and along the other to malignant adenoma, is so insensible that I am persuaded that we are confronted with a series of responses not different in kind, but only differing in degree. That there is some connection between functional capacity and this hyperplastic response is suggested by the frequency of the papilliferous change and the period at which it takes place in lateral aberrant thyroids, the tissue of which is probably imperfectly developed and therefore of low functional value, and by the higher incidence of malignant adenoma in centres of endemic goitre where the affected glands are sub-efficient.

Although carcinoma of the thyroid is rare in comparison with its occurrence in some other organs such as the breast and the uterus, and therefore material for research is scarce, and although in the early stages the difficulties of drawing the line dividing innocency from malignancy are so great, this organ presents some unusual advantages in the attempt to unravel the difficulties. Among these advantages is the extreme responsiveness of the thyroid gland to the demands of the organism in human beings, and the wide range of these demands (varying from puberty to grave psychic disturbance, and possibly endocrine imbalance), while in animals—as in brook trout—the gland may be subjected to experimental observation. This responsiveness is such that deviations from the normal are quickly recognized and are reflected in the histology of the gland. Apart from malignancy, many of its diseases are, for the moment, dealt with surgically, and therefore much material is available for the investigation of the histology of its very varied pathological conditions, as well as of its chemical and biological activities. This gives opportunity to correlate the work on innocent and malignant tumours of the thyroid gland with that of Lenthal Cheatle<sup>21</sup> on "The Primary Tumour in Breast Carcinoma", of Sampson Handley<sup>22</sup> on



"The Menace of the Papilloma", and of Robert Muir<sup>26</sup> on "The Intra-epithelial Growth of Carcinoma" (preceding invasion of the basement membrane). It should also enable us clinically to avail ourselves of the work of Marine and his co-workers, of McCarrison, Mellanby, and others, who have made such valuable contributions to the animal experimentation side of this problem.

### SUMMARY.

#### Pathological.—

1. In our cases there are three types of cancer of the thyroid: scirrhus, papilliferous adenocarcinoma, and malignant adenoma.

2. Scirrhus does not differ from the same disease occurring elsewhere in the body.

3. In the thyroid gland epithelial proliferation is the characteristic response to stimulation. The proliferation may resolve spontaneously or under treatment; it may form a benign tumour, or it may form a tumour which invades and disseminates. These stages merge into one another by insensible gradations. These histological gradations cause difficulty in deciding just when a tumour has become malignant.

4. Proliferation of thyroid epithelium may be papilliferous or follicular in type. Papilliferous adenocarcinoma may be an ultimate result of the former, malignant adenoma of the latter type.

5. It is suggested that the cause of carcinoma is stimulation, which may be normal and affecting tissue which is sub-efficient, or so excessive in degree as to be abnormal and amount to irritation.

6. Although in cases of carcinoma of the thyroid specific glandular structure is frequently retained both in the parent growth and in the metastases, the essential character of the disease conforms to the same laws observed in carcinoma of other glandular organs, and its cause is possibly the reaction of the glandular epithelium to irritation as suggested by other workers in, for example, carcinoma of the breast.

#### Clinical.—

1. A nodule in a thyroid gland should not be regarded as of no importance, and early changes in the signs or symptoms associated with it should induce the practitioner to investigate the cause of these changes.

2. Histological examination should be made in every case of removal of thyroid tissue, and sections should be taken from different areas.

3. Advanced cases should not be regarded as hopeless. When the condition of the patient justifies it, as much of the tumour should be removed as possible, and then X-ray treatment commenced. By this means comfort is given, and life is sometimes prolonged to a surprising extent.

The sections shown in this article have been cut and the microphotographs made by Miss M. Vaughan of the Surgical Professorial Unit, St. Bartholomew's Hospital. Some of the illustrations are appearing in the new edition of Choyce's *System of Surgery*, and these are reproduced by mutual agreement.

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## THE PATHOLOGY AND TREATMENT OF TUMOURS OF THE CAROTID BODY.

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THE carotid body was first described by Neubauer in 1786, although Haller<sup>1</sup> had mentioned its presence many years previously. Since then many different names have been given to this apparently insignificant structure, according to whether it has been thought to be an endocrine gland or a ganglion. Following the work of Kohn<sup>2</sup> it was generally accepted that the carotid body was derived from embryonic ganglion cells of the sympathetic plexus, and was therefore included among the chromophil tissues. However,

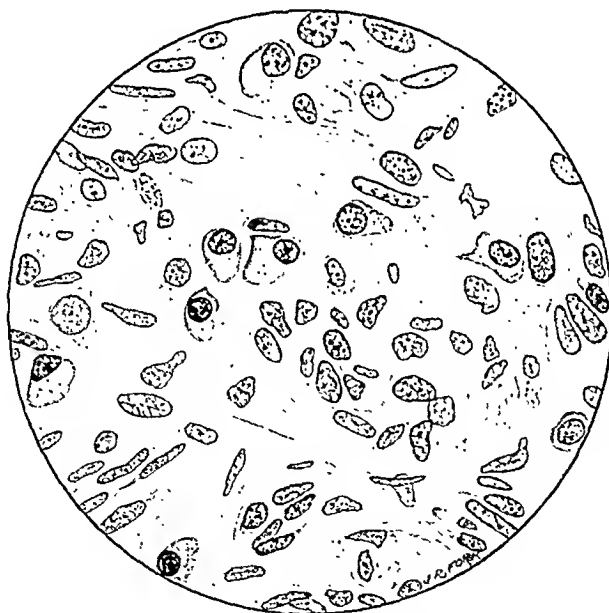


FIG. 118.—Section of normal carotid body, to show various types of cells which are met with in this structure. Attention is specially drawn to the large cells with a darkly stained nucleus situated eccentrically and finely granular and vacuolated cytoplasm. ( $\times 600$ .)

de Castro<sup>3</sup> has recently produced evidence to show that the structure does not contain adrenalin; he suggests that the carotid body is related in some way to the carotid sinus.

I have dissected out a number of normal carotid bodies in the cadaver and examined them microscopically (*Fig. 118*). They are small pinkish bodies

situated in the middle of, or just deep to, the bifurcation of the common carotid artery on each side. The average size is  $\frac{3}{16}$  in. long,  $\frac{1}{8}$  in. broad, and  $\frac{1}{16}$  in. thick. They have a definite capsule, and are attached to the bifurcation of the common carotid artery by a short fibrous pedicle. A branch of the glossopharyngeal nerve can be traced to this region. From the connective-tissue capsule fibrous septa enter and divide the structure into lobules, which are again subdivided by smaller trabeculae of the same connective-tissue network. The organ is richly supplied with blood-vessels and nerves. The cells may be divided into two groups:—

*Group A.*—The majority are large polyhedral cells, rich in cytoplasm, which is finely granular and poorly staining. These cells occasionally form a syncytium. The nuclei are large, round or oval, somewhat excentrically placed, rich in chromatin, and show a distinct nuclear membrane. Other cells, resembling these, are found; they are slightly larger and their cytoplasm, which is less granular, contains small vacuoles. The more vacuolated the cytoplasm, the less granular it becomes. These vacuolated cells are apparently derived from the granular cells.

*Group B.*—Many endothelial-like cells are to be found, particularly around the periphery of the lobules. They are small or medium-sized, have little cytoplasm and deeply-staining nuclei, which are oval or spindle-shaped. Occasional eosinophil tissue-cells are to be seen scattered about the structure, and fibroblasts are present in the connective-tissue framework.

### CASES OF TUMOUR OF THE CAROTID BODY.

*Case 1.*—The patient, a carpenter, age 30, was admitted to St. Thomas's Hospital in February, 1928, under the care of Mr. Max Page.

*HISTORY.*—For four years the patient had suffered from attacks of sharp pain starting in the left side of the head, shooting down to the neck and to the left shoulder. In these attacks he fell to the ground, but did not think he lost consciousness; in a few minutes he recovered and was able to get up and to continue his work.

*ON EXAMINATION.*—There was a swelling about the size of a large walnut in line with the left carotid vessels, and situated at the bifurcation of the common vessel. It was fairly mobile laterally, but fixed vertically. The lump was firm in consistency and gave a transmitted pulsation from the main vessels. On firm pressure on the lump the man's pulse became slow and feeble. Dr. M. A. Cassidy obtained tracings which show the result graphically of the effect of pressure. No definite diagnosis was made.

*OPERATION (Feb. 3).*—A 3-in. incision was made along the anterior border of the left sternomastoid, and the tumour was exposed. The internal jugular vein appeared greatly distended, and it was necessary to divide it below the level of the tumour and strip it up before the relationships of the latter could be made out. The tumour was then found to be vascular and firmly adherent to the carotid vessels, so that resection of the latter was clearly necessary in order to effect its extirpation. Considering the severity and danger of the man's condition, it was decided that in this case extirpation was justifiable, even in the face of the great operative risk. After resection of a length of the jugular vein, the common carotid artery was divided half an inch below the tumour and, together with the latter, was stripped away from the underlying structures; the vagus nerve was recognized and left intact. Part of the sympathetic chain was adherent to the posterior aspect of the tumour and had to be resected. Post-operative progress was uneventful, except for transient aphonia. A slight enophthalmos and definite miosis were noted

the day after operation and have persisted. The patient remains in perfect health at the present time and free from symptoms.

**MACROSCOPIC APPEARANCE.**—The tumour was ovoid and measured 4.5 by 3 cm. It was definitely encapsulated, and had a firm homogeneous structure on section; it was pale pink in colour. It was traversed by part of the common and the internal and external carotid arteries, to which it was closely adherent.

**MICROSCOPIC APPEARANCE (Fig. 119).**—The tumour was malignant (Professor L. S. Dudgeon).

**Commentary.**—I have found over a hundred cases of new growth of the carotid body recorded, but in only three of these was the tumour associated with syncopal attacks. In the light of recent physiological knowledge of the function of the carotid sinus, the case recorded above is of great interest. De Castro<sup>3</sup> and Heymans<sup>4</sup> believe that the carotid body is closely related to this

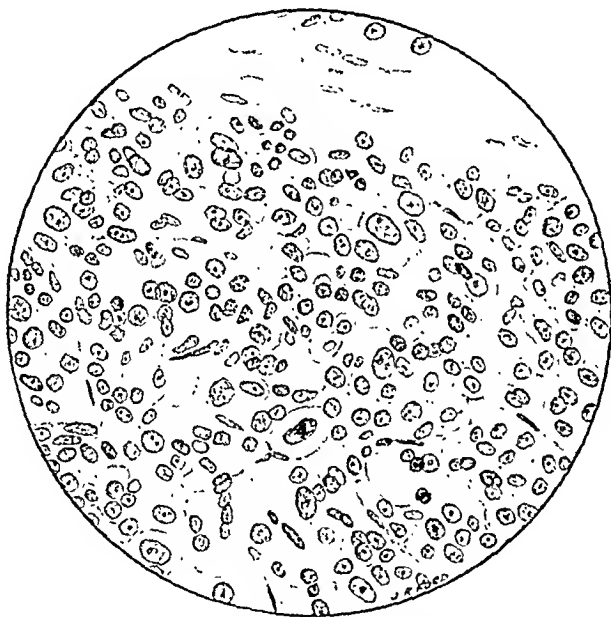


FIG. 119.—Case 1. Section of a malignant growth arising in the carotid body. This tumour, which is of the renal hypernephroma type, had invaded its capsule and extended into the lymphatic vessels. Note the appearance of some very large cells in the figure. ( $\times 270$ .)

structure in function. In the physiological sense, the carotid sinus is a specially innervated part of the vessels in the neighbourhood of the bifurcation of the common carotid artery into its branches, i.e., the carotid bulb. This sinus and the carotid body are supplied by the glossopharyngeal nerve. By perfusion experiments it has been shown that stimulation of the carotid sinus, electrically or mechanically, provokes a combined reflex of cardiac inhibition and fall of blood-pressure, just as does stimulation of the central end of the depressor nerve. It is well recognized that pressure over this region of the neck causes slowing of the heart and syncopal attacks. Hering<sup>5</sup> has shown that this is not due to pressure on the vagus, as was previously supposed, but that it is due to pressure on this sinus. It is therefore easily

understood why pressure on the tumour produced the attacks in this patient. It is more difficult, however, to explain why the symptom is not produced in all cases.

*Case 2.*—A male patient, age 67, was admitted on June 10, 1929, to St. Thomas's Hospital, under the care of Sir Percy Sargent.

*HISTORY.*—The patient complained of a swelling on the right side of the neck of one year's duration. Hoarseness of the voice and some difficulty in swallowing had been noticed for a few months. He was otherwise healthy and did not complain of any pain.

*ON EXAMINATION.*—A firm, smooth tumour, of  $2\frac{1}{2}$  in. diameter, was seen on the right side of the neck at the level of the thyroid cartilage and in the line of the great vessels. It was not attached to the skin, but moved on swallowing and was apparently of thyroid origin. The trachea was displaced one inch to the left. The

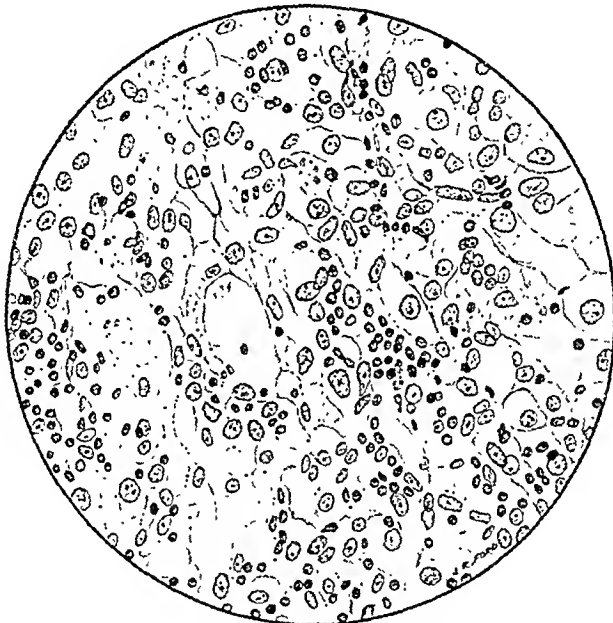


FIG. 120.—*Case 2.* Section of malignant growth arising in the carotid body which had invaded the sternomastoid muscle. ( $\times 270$ .)

tumour was only slightly movable laterally, but was fixed vertically. Laryngoscopy revealed no abnormality.

*OPERATION (June 19).*—The tumour was found to be very adherent. The case was regarded as one of inoperable carcinoma of the thyroid gland, and a portion was removed for microscopy.

*MICROSCOPIC APPEARANCE (Fig. 120).*—Malignant tumour of the carotid body (Professor L. S. Dudgeon).

*IRRADIATION.*—On Aug. 26 the patient was re-admitted; the tumour had increased slightly since the previous admission. Thirty radium needles of 1 mgrm. each, screened by 0.5 mm. of platinum, were inserted into and around the growth through skin punctures and retained for a period of fourteen days. The tumour thus received an irradiation of 10,080 mgrm.-hours. A slight radium burn occurred. Following the irradiation the tumour decreased in size and swallowing became easier. The hoarseness of the voice persisted. On Oct. 27, 1930, fourteen months after the radium treatment, the tumour had reached its original size and was growing actively towards the mid-line. Hoarseness had increased and pain in the neck had been

present for three months. The tumour was still unattached to the skin, but was more fixed to the muscles. Slight movement still occurred on swallowing. The tumour was irradiated with 10 needles of 1 mgrm. each with the same screenage as before. A severe general and local reaction occurred, which necessitated the removal of the radium after eleven days' irradiation. On this occasion the radium treatment failed to have any beneficial effect. The patient was seen recently. He was very much weaker, in severe pain, and the tumour was growing rapidly. No evidence of any metastases could be found clinically.

**Commentary.**—The following points are of interest in this case: (1) The tumour on clinical examination appeared to be of thyroid origin. As the tumour was more actively growing towards the mid-line, it invaded the thyroid structures before becoming fixed to the other structures of the neck, and thus movement on swallowing occurred. As the growth progressed, the fixity became more general and movement on swallowing thus gradually diminished. (2) The effect of radium in this case was without permanent result. (3) Although the tumour was malignant, no evidences of glandular or general metastases were found clinically.

*Case 3.*—Mrs. G. W., age 41, a window-dresser, complained of a swelling on the left side of the neck, which had been present nine years. The tumour was painless and caused no symptoms. She was healthy.

**ON EXAMINATION.**—A firm, smooth tumour was to be seen in the left side of the neck, just below the angle of the mandible. It measured  $1\frac{1}{2}$  by 1 in., and lay in the line of the carotid vessels. The tumour was unattached to skin and was freely mobile from side to side, but fixed vertically. There were no other abnormal physical signs.

**OPERATION** (Feb. 27, 1929).—The tumour was exposed by Sir Percy Sargent through an oblique incision on the left side of the neck. It was completely encapsulated, and closely attached to the bifurcation of the left common carotid artery. The tumour was dissected free from the vessels and removed entirely without damage to nerves or main vessels.

**MACROSCOPIC APPEARANCE.**—The tumour was pinkish in colour, homogeneous, and completely encapsulated.

**MICROSCOPIC APPEARANCE.**—A non-malignant tumour of the carotid body (Professor L. S. Dudgeon).

**SUBSEQUENT HISTORY.**—The patient made an uninterrupted recovery. She was examined recently and found to be entirely free from symptoms or recurrence.

*Case 4.*—A woman, age 70, had noticed a swelling on the left side of the neck for four years. It was painless and caused no symptoms, but recently had grown larger. The tumour measured 1 by  $\frac{3}{4}$  in., situated at the bifurcation of the common carotid artery. It was thought to be an enlarged gland.

**OPERATION** (July, 1930).—The tumour was removed by Sir Percy Sargent without damage to nerves or main vessels. The patient made an uninterrupted recovery and there has been no recurrence.

**MACROSCOPIC APPEARANCE.**—The tumour was pink in colour, encapsulated, and had a homogeneous structure.

**MICROSCOPIC APPEARANCE.**—The tumour showed no evidence of malignancy (Professor L. S. Dudgeon).

## PATHOLOGY, DIAGNOSIS, AND TREATMENT.

### **Pathology.**—

**MACROSCOPIC APPEARANCES.**—The tumours which were removed were pinkish in colour and encapsulated. On dividing them the structure was

homogeneous and closely resembled that of a foetal adenoma of the thyroid. In *Case 1* portions of the common, internal, and external carotids were embedded in the centre of the tumour. The malignant tumour in *Case 2* was irremovable.

**MICROSCOPIC APPEARANCES.**—I examined the microscopical sections of these four cases, and found that they were composed principally of cells resembling those of Group A (*see p. 115*) of the normal carotid body.

*Non-malignant Tumours (Cases 3 and 4).*—The large cells were arranged in more definite clusters than in the normal structure. These cell-groups were surrounded by young fibrous tissue. The cells had the same polyhedral form, and the cytoplasm was finely granular and stained faintly. The nuclei were rich in chromatin and had distinct nuclear membranes. The cells were slightly larger and vacuolation was more marked and blood-vessels were more abundant than in the normal structure. The capsule was not invaded. Endothelial cells were present and also eosinophil and plasma cells. In places there was but little fibrous tissue present and the cells were in sheets.

*Malignant Tumours (Cases 1 and 2).*—The cells in these cases were in masses, without separating fibroblasts. *Case 2* was obviously the more malignant, not only clinically, but also from the microscopical point of view. Definite invasion of the capsule was present in both cases, but in *Case 2* the invasion had spread to the surrounding adherent muscle, which also showed a myositis.

The cells were much larger than in the normal organ or in the benign tumours. Vacuolation was gross in *Case 2*, particularly in the centre of the tumour, but the nuclei retained their definite and well-staining properties, and nucleoli were occasionally present. Mitotic figures could be seen. In many places the loose cellular structure had broken down, and hæmorrhages were present. At the periphery of the tumour the vacuolation was less conspicuous and endothelial cells were present in abundance, particularly in those regions where the tumour was invading its capsule and adherent muscle. The blood sinuses were large. It is noteworthy that the number of eosinophil cells was in excess of that normally found in blood sinuses.

**Diagnosis.**—The definite diagnosis of a fixed malignant carotid body tumour before microscopy would appear impossible. In the past, simple tumours have rarely been given a correct pre-operative diagnosis, mainly because of their rarity. Considering the recorded cases and those mentioned in this paper, the diagnosis of simple or early malignant tumours of the carotid body would depend upon the following characteristics: (1) The tumour is slow-growing. Therefore a large tumour in the neck, of short duration, is unlikely to have arisen from the carotid body, unless there is obvious evidence of malignancy. (2) The situation at the bifurcation of the common carotid artery. (3) Free mobility laterally, but not in the line of the vessels. This lateral mobility diminishes when the tumour is malignant. (4) Practically always unilateral and equally common in both sexes. (5) No fixation to the skin. (6) Transmitted pulsation. (7) Painless and not tender to palpation. (8) The tumour may in rare instances cause syneopical attacks. (9) When the tumour is large, pressure symptoms may occur, e.g. aphonia, dysphagia, dyspnoea, etc. (10) Metastases have not been found, even in malignant cases.



Bevan and McCarthy<sup>6</sup> analysed most of the recorded cases. A few of their figures will be quoted: (1) The majority of cases occur between the ages of 30 and 40, and practically 70 per cent between 30 and 60. The average age is 41.7 years. (2) The average duration of the tumour before consultation is  $7\frac{1}{6}$  years. (3) Of carotid body tumours 80 per cent are benign.

**Treatment.**—In considering the treatment of these tumours there are certain facts to be borne in mind: 80 per cent of carotid body tumours are said to be non-malignant; 70 per cent occur between the ages of 30 and 60. The risk attached to resection of portions of the carotid arteries together with the bifurcation of the common vessel is very considerable, and particularly after the age of 40. The mortality is stated to be 30 per cent, and hemiplegic signs occur in some of the patients who survive.<sup>6,7</sup> Therefore, should resection of the vessels be necessary, the operation is fraught with greater danger in the majority of cases. Thus, it would appear that an exploratory operation should be undertaken in all cases clinically operable. If the tumour proves removable without resection of the internal and common carotid arteries, this procedure should be adopted. A tumour which is found to be irremovable except by resection of portions of the vessels mentioned should be left, unless the condition of the patient warrants the severe operative risk. Syncopal attacks definitely due to the tumour, or evidence of malignancy as shown by diminished lateral mobility or microscopy, would be the chief indications for this procedure. The age of the patient, and particularly the condition of the arteries, should also be taken into account.

Radiotherapy has not been used in a sufficiently large number of cases to enable any definite idea to be formed of its value. In one of the malignant cases recorded, radium treatment was followed by no permanent benefit, and Birman<sup>8</sup> observed the same result. Radium should therefore be reserved only for those malignant cases which are inoperable, as its beneficial effect is too uncertain.

I am indebted to Sir Percy Sargent and Mr. Max Page for permission to publish these cases, and also to Professor L. S. Dudgeon for much help and advice.

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## THE OPERATIVE TREATMENT OF POTT'S PARAPLEGIA.

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My object in writing this paper is to advocate early decompression for Pott's paraplegia in adults. The decompression may take the form of laminectomy, costotransversectomy, or both; but, whatever the method of decompression, I regard stabilization by bone-grafting of the diseased part of the spine as an essential complement.

This paper refers particularly, though not exclusively, to adults, for in my experience children with spinal caries under modern treatment very rarely suffer from severe paraplegia. Probably this is because we are learning to keep them at rest sufficiently long, with the spinal column relieved from strain and metabolism stimulated by open-air conditions. The surgical rest will almost always quickly cure the earlier milder paraplegia, and time, if sufficiently prolonged, prevent the development of late paraplegia—for this disaster has been usually associated with insidious persistent caries after the child had been allowed to get up. On the whole, I would say of Pott's paraplegia that in children it can as a rule be avoided or quickly cured by conservative treatment, but that in adults operation is generally indicated.

There has recently appeared a monograph by Madame Sorrel<sup>1</sup> in which conservative treatment of Pott's paraplegia is advocated. Madame Sorrel does not advocate operation because: (1) In early cases she prefers to continue conservative treatment in the expectation that in the majority the paraplegia will sooner or later disappear; (2) In late cases her experience is that the great majority do not get well whether anything is done or not.

Nevertheless I am at present a whole-hearted believer in operation on patients suffering from severe paraplegia, whether it has appeared early or late. I have long been convinced of the neurological advantage of early operation. Simultaneous bone-grafting has altogether removed the great drawback of laminectomy, and the results have been most encouraging. I feel that the combination of grafting with laminectomy and the stabilization so achieved make a great, and indeed a critical, difference to the arguments for and against decompression. The cure of Pott's disease depends on two things: good general health, and (this is our immediate concern) restored spinal stability. Caries undermines the stability of the spinal column, the resultant instability in its turn favours further erosion, and a very vicious circle is thus set up. Now laminectomy has been discountenanced because it produces a further structural defect, and this is in truth a drawback which only very strong indications could overcome. But if, by combining grafting with laminectomy we can increase, not diminish, the stability of the spine, this drawback exists no longer, and the whole outlook is changed.

I believe that decompression is indicated in many of the early cases in adults, both because some do not clear up at all without operation, and because in many of the others there is a long period of compression which is dangerous and harmful to the cord. Also this long-continued paraplegia is harmful and most distressing to the patient, and this at a time when restoration of his lost vitality is an urgent need. Instead of peace and progress, the patient experiences painful muscular spasms, is with difficulty protected from pressure sores, and, if the sphincters are affected, from ascending genito-urinary infection. Pott described this condition as "most miserable to endure, most pitiable to see".

As has been said already, in my part of the world Pott's paraplegia in children is now becoming extremely uncommon. I have had hundreds of children suffering from spinal caries under my care, and in the last ten years remember only two or three cases of paraplegia. Indeed, I have only twice had to do a laminectomy for Pott's paraplegia in a child. So what I have to say concerns more particularly Pott's paraplegia in adults.

In my experience paraplegia generally comes on in the first few months of the disease, and in these cases the pressure is almost always from abscess, granulation tissue, débris, etc., in front of the theca. If the paraplegia advances quickly, or if it advances progressively during immobilization, I operate to relieve compression by costotransversectomy or laminectomy, or both. In addition I do a twin graft fixation; after laminectomy these grafts bridge the opening and more than counteract the local loss of laminae and spines. In effect a mobile chain of laminae is replaced by a pair of strong grafts which hold the diseased area of spine much better than any external splint can do. The grafts must be stout, for the strain on them is great; the stresses are largely concentrated at the level of the disease, and it is here that the grafts are liable to fracture, or to show discontinuity as a result of local osteoclasia from overstrain of the bone-cell-spicule units.

Early decompression is, then, I believe, indicated to protect the cord from prolonged pressure and the patient from the complications of paraplegia: and grafting, which leads to complete local immobilization of the spine, is indicated to protect the spinal column from further erosion and to promote healing of the lesion.

There are those who think that laminectomy is not helpful in Pott's paraplegia because the pressure almost always comes from in front: they hold that the cord is not compressed against the laminae but strangulated by being tied down against this anterior pressure by the intervertebral nerves and their dural attachments. This view appears to me theoretically unsound, and it is contradicted by my experience both in operative findings and in the results of laminectomy. *Case 1* is particularly convincing. Not only was the complete paraplegia very rapidly relieved by laminectomy, but when the paraplegia recurred it proved to be due to the pushing of the theca *backwards through the laminectomy opening* and the compression of the cord against the lower edge of the opening. This was a case of rapidly advancing paraplegia; and yet the theca not only was first compressed against the laminae, but after this pressure was relieved by laminectomy the theca was gradually pushed even further backwards and bulged beyond the level of the laminae

(see Figs. 121, 122). In this case without doubt it was the laminae and not the nervous and dural attachments of the theca which opposed its backward displacement by pathological pressure from in front. This is, I believe, the rule whenever there is gross extrathecal pressure from in front.

Now for a moment to consider spinal caeries apart from the paralysis: in 1923 I advocated operative fixation for all adults suffering from spinal caeries where no contra-indication existed,<sup>2</sup> and I have learnt to value operative fixation more and more during the seven years since then. Operative spinal fixation in good hands is almost free from risk, and there is little or no disturbance of the lesion if a technique is adopted which maintains immobilization of the spine before, during, and after the operation. I believe that one should graft early, but not until three conditions have been awaited. The patient should be settled comfortably on his frame or plaster bed, and past the period of active ill health; further, the number of vertebrae involved by the disease should be known so that the length and site of the grafts may be correctly judged. Now by the time paraplegia (other than the transient form) has developed, the extent of the disease has become clear. But grafting shuts the door on laminectomy. I have once done a hemilaminectomy on a case in which two grafts had been applied deep down on each side of the spinous processes. This operation was so difficult as to be dangerous, for a thick continuous layer of bone replaced the chain of laminae. Clearly, then, it is wise in cases of severe paraplegia to do an adequate laminectomy before putting in grafts.

In the last ten years I have had under my care 12 cases of Pott's paraplegia on whom I have operated for relief of pressure on the cord. The operations have been as follows: costotransversectomy and graft 2, laminectomy and graft 5, laminectomy and graft+costotransversectomy 4, laminectomy alone on a case which I had previously grafted 1. In one case where a graft fractured after a fall I re-grafted and at the same time re-opened the costotransversectomy and evacuated a further collection of debris, so that there have been altogether thirteen operations. In most cases the operative interference is completed at one stage, but in three of the cases where a laminectomy, graft, and costotransversectomy have been done, the laminectomy and graft has been at one stage and the costotransversectomy at a second. There have been two deaths, both in late and clearly almost hopeless cases. In one, that of an old lady, death occurred within twenty-four hours from shock. The other case, a man of 34, died about eighteen months later in coma. One case of laminectomy, graft, and costotransversectomy has been done too recently to allow of any progress report other than that the patient was undisturbed by the operation. Of the other 9 cases, recovery, partial (4) or complete (5), has occurred. It is noteworthy as regards two of the cases with complete recovery that the paraplegia had occurred late on in the second year of the disease. Further, of the four partial recoveries, two are still improving progressively and seem likely to recover completely.

There are, then, three operations to be considered: (1) Costotransversectomy; (2) Laminectomy; (3) Grafting. But even if all three are done (in one-third of my cases) there need only be two stages, for the grafting is a supplementary part of the laminectomy operation. The first question is

whether to do a costotransversectomy or a laminectomy. If this doubt arises, it is often wise to do both; then it remains to decide which should be done first. The argument in favour of doing the laminectomy first is the advantage of the earlier immobilization following the graft, for in Pott's disease with paraplegia one is especially anxious to limit the local destruction and the piling up of the 'granuloma-débris dump' as quickly and decisively as possible. On the other hand, costotransversectomy may eliminate the need for laminectomy. But in considering this one should remember the occasional occurrence of a tuberculous sinus after a costotransversectomy through tuberculous infection of the wound track. (*See Case 6*, in which a sinus developed four to five weeks after operation.) Fortunately such a sinus takes several weeks to appear, so there is time during which one can generally get guidance as to the need for laminectomy, and then carry it out as part of the graft operation after the costotransversectomy and before the time when a sinus might develop. Such a sinus, though uninfected by pyogenic organisms and several inches away, would make one much less happy in performing the laminectomy-graft operation.

I favour doing the costotransversectomy first if X rays show a spherical, or fusiform but nearly spherical, prevertebral abscess, for this appearance suggests that there is still a tight and localized distension of the prevertebral fascia by *débris* which, unable to escape upwards or downwards, has found its way backwards. In such cases I generally do a costotransversectomy first, then a fortnight or so later a graft, and unless the paraplegia is improving I do a laminectomy also at this second operation. It is occasionally advisable to evacuate the *débris* a second time if the clinical state and radiograms indicate the need (*see Cases 6 and 9*).

Then with regard to the second group of cases in which paraplegia comes on late in the history of Pott's disease, Madame Sorrel's view is that nothing will cure 90 per cent of these, and therefore she counsels inaction. Admittedly these late cases are much less hopeful. They are less likely to get over the tuberculous disease, and the local cause of the compression is less often uncomplicated extrathecal compression. It appears to be impossible at present to pick out with any certainty those patients whom operation will relieve or cure; probably it is still harder to distinguish those in whom prolonged compression has led to permanent changes in the cord or in whom the paraplegia is due to widespread thickening of the dura. My own view is that one should frankly explain the situation, then advise operation, but only perform it if the patient desires it, being fully conscious of the uncertainty of the outcome. I would advise operation even for those who from age or lack of vitality seem little likely ultimately to recover from the tuberculous disease, for I would myself so far prefer a recovery from paralysis, a period of symbiosis with tubercle bacilli, and the milder death of generalized tuberculosis, to continued paraplegia with all its discomforts and an end no less inevitable and probably preceded by sores and urinary sepsis. But I am much more hopeful than Madame Sorrel as to the percentage of late cases which can be cured or relieved. *Case 8* shows that operation, even in a very late case, may be worth while. Fortunately I see few of these late paraplegias, probably because I work in a district in which almost all the cases

of spinal cases come early to the orthopaedic hospital and get prolonged general treatment and careful after-care. Also almost all the adults are grafted as soon as the indications are satisfied, and I feel very strongly that in adults the strong double graft which I use is a very effective internal splint, which checks erosion and debris production, limits the extension of the disease, and thus minimizes the chances of paraplegia.

### CASE REPORTS.

*Case 1.*—Male, age 19. Admitted to the Wingfield Orthopaedic Hospital on Jan. 12, 1921.

**HISTORY.**—Kyphosis in dorsal region present for an indefinite period. Onset of loss of power in both legs commencing three weeks before admission. Patient walked up to out-patients' department with spastic gait.

**X RAY.**—Bodies of D.6, 7, and 8 largely destroyed. Abscess shadow.

Jan. 12, 1921.—Voluntary power: right and left legs nil. Spasms: right and left legs ++. Sensation: light touch lost below 8th dorsal. Sphincters: not

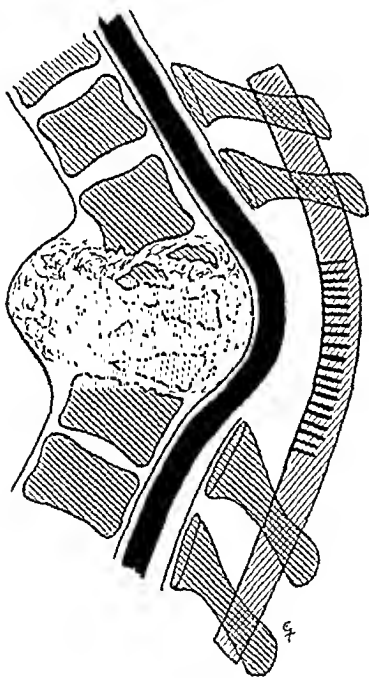


FIG. 121.—*Case 1.* Condition at the end of the first operation (Jan. 28, 1921). Graft shown in red, with saw cuts to allow flexion.

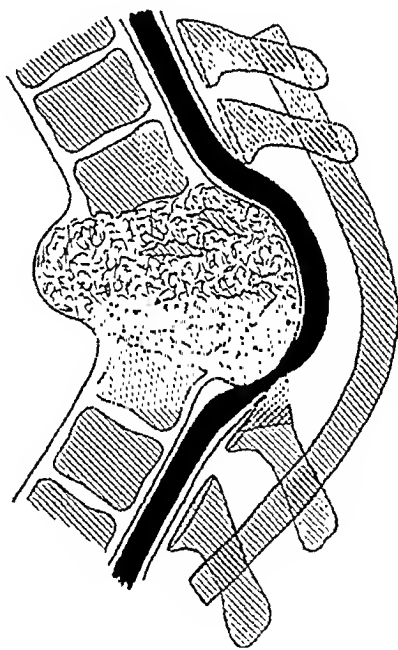


FIG. 122.—*Case 1.* Compression found at second operation (June 12, 1921). Area of further laminectomy shown cross-hatched in red.

affected. Reflexes: abdominals —; knee-jerks +++; ankle-jerks +++; clonus, present; plantar, extensor (all right and left). Wassermann: negative.

Despite immobilization on spinal frame the paralysis deepened, and became complete, motor, sensory, and sphincter.

Jan. 28, 1921.—*Laminectomy and graft.* D.6, 7, and 8 spines and laminae removed. Dura pushed through opening at once and began to pulsate well. Prominence palpable in front of cord. This had clearly compressed the cord against the laminae. Tibial graft fitted to bridge gap from two spines above to two

spines below. Many transverse cuts to make it fit. Immediate result: Sensation and muscular control of the lower limbs recovered the same evening; but this improvement was not maintained, and in two or three weeks there was complete sensory and motor paralysis and incontinence.

June 12.—*Enlargement downwards of laminectomy opening* without damage to graft. Graft and membranes inspected. The theca was found projecting backwards through the laminectomy opening and pushed against the lamina at the lower edge of this opening. I found it possible to remove enough of this lamina by undercutting the graft.

*Fig. 121* indicates the state of affairs at the end of the first decompression. *Fig. 122* shows the compression found at the second operation owing to increased accumulation in front of the theca. It was noteworthy that the graft was rounded, covered with a vascular fibrous membrane, and had retained its arch; it appeared to form an extremely strong and fully adequate internal splint.

July 14.—Incontinence of urine and faeces ceased.

Dec. 30.—Complete recovery of sensation and sphincter control; partial of voluntary power.

Dec. 12, 1922.—Getting up in spinal support and calipers and learning to walk.

March 21, 1923.—Great improvement in walking.

April 30.—Discharged.

March 3, 1926.—Very well indeed.

This patient is now perfectly well and active. There is no trace of paraplegia left. He has been at work for years.

**Comment.**—This was a case in which the relief of paraplegia was dramatic in its swiftness and completeness. The onset of paralysis was rapid and total, and the recovery of sensation and movement in the lower limbs followed operation immediately. A relapse followed owing to enlargement of the mass in front, and an insufficient area of decompression. Relief was again obtained, though not so swiftly, by a second operation and removal of more bone at the lower angle of the laminectomy opening. The final result has been complete restoration of function and a normal working life.

There are several points of great interest in this case:—

1. It was the loss of power and awkwardness in his legs that brought the patient to hospital.

2. The loss was rapidly progressive from the time when the patient was first seen until the date of operation, although he was kept completely lying down until he could be admitted and immobilized on a spinal frame.

3. Laminectomy was done early, and the relief of paralysis was complete and immediate.

4. But it recurred, and, as was found at the second operation, was due to further accumulation of debris between the centra and the theca and to crushing of the theca against the lamina as shown in *Fig. 122*.

5. The second operation was not done early, and the recovery was delayed, though ultimately complete.

6. I would add that nowadays I should have done a costotransversectomy before or within a week or two of the laminectomy and graft, and I feel sure that this would have prevented the return of the paralysis.

It was difficult to decide whether 'to go in again' early or not, and I am confident that a quicker recovery would have followed either costotransversectomy and evacuation of the abscess or an earlier extension of the laminectomy.

The second operation demonstrated the possibility of reaching the theca to one side of a graft bridging a laminectomy. This is made easier when the graft or grafts are not bedded deeply. It also enabled me to see how very strong the graft was—a thick rounded living bridge, covering, protecting, and completely immobilizing the area of disease and laminectomy.

*Case No. 2.*—Male, age 26. Admitted to the Wingfield Orthopædic Hospital on Jan. 24, 1921.

*HISTORY.*—Injury to back in 1918. Pain in back ever since; worse since July, 1920. Pain in abdomen in August, 1920. Numbness of legs and later loss of power in September, 1920.

*X RAY.*—Shows considerable destruction of bodies of D.8 and 9, with abscess shadow.

Voluntary power: right and left legs, nil. Spasm: right and left legs ++. Sensation: light touch +; pinprick diminished below 8th dorsal segment. Incontinence: difficulting in starting and scarcely aware of act for both urine and fæces. Reflexes: knee-jerks ++; ankle-jerks +; clonus (ankle) +; plantar, extensor (all right and left). General condition very poor.

March 14, 1921.—*Laminectomy.* Laminae of D.7, 8, and 9 removed. Pulsation absent till D.7 laminae removed, then returned at once. Carious material found and removed from posterior surface of body of D.8, which appeared widely destroyed, and showed considerable respiratory movement. In view of poor general condition, graft was postponed.

May 27.—All muscles acting strongly in right and left legs. Very few spasms.

Oct. 10.—Still complaining of pain across chest and to back. (This persisted until graft.)

Nov. 21.—*Graft.* Tibial graft from D.4 to D.9 inclusive, bridging laminectomy gap. Multiple transverse cuts to adapt graft to kyphos.

August, 1922 (approx.).—Discharged wearing spinal support.

*N.B.*—*Discharged unduly early as he could not stay in hospital owing to very bad home conditions of wife and family.*

Feb. 25–Oct. 22, 1925.—Re-admitted and put on frame for pain in back and chest and general ill health. Very bad home conditions. No paraplegic symptoms or signs.

*X RAYS.*—Showed discontinuity of graft opposite D.9 and abscess shadow to right of D. 8, 9, and 10. Discharged better and wearing support.

*N.B.*—*In my present practice I should have patched and reinforced the graft.*

Feb. 14, 1929.—Re-admitted for pain in back and chest, weakness in legs, and general ill health. No definite clinical signs of paraplegia.

*X RAY.*—Discontinuity of graft, and abscess shadow.

March 5.—Definite paraplegic signs and tendency to incontinence. On plaster bed for operation.

April 22.—*Operation.* (1) Two fresh tibial grafts cut and laid alongside old ones on each side over whole length of arca; (2) Costotransversectomy on right side begun, but abandoned incomplete owing to condition of patient.

May 20.—No improvement. Voluntary power: right and left legs, nil. Spasm: right and left legs +++. Sensation: below 8th segmental level —. Incontinence: automatic bladder and rectal action with no voluntary control. Reflexes: knee-jerks + + +; ankle-jerks +; plantar, extensor; clonus (ankle) +; abdominals — (all right and left).

May 21.—*Costotransversectomy.* Vertebral end of rib and transverse process of corresponding vertebra opposite kyphos on right side excised and much bone sand curetted away. No large sequestra. No pus.

Feb. 17, 1930.—Soundly healed. General condition excellent. Voluntary power: right and left legs, complete. Spasms: right and left legs, nil. Sensation: diminished (but present) below 8th dorsal segmental area. Incontinence: nil. Reflexes: abdominals —; knee-jerks + + +; ankle-jerks + + +; clonus (tendency, right), left, nil; plantar, extensor (all right and left).



**Comment.**—Here the onset of paralysis was gradual, but had become practically total when the patient was admitted to hospital some five months after the first appearance of symptoms. Laminectomy and later graft resulted in restoration of function sufficient for discharge in seventeen months. His home circumstances were very difficult, and the strain to which he was unavoidably exposed led to recrudescence of activity in his spinal focus. Finally paraplegia recurred eight years after his original laminectomy. For this he was first re-grafted, and as this with its necessary accompaniment of complete immobilization produced no improvement in his condition, costo-transversectomy was performed and much tuberculous material thereby given an exit. This has resulted, nine months later, in practically complete restoration of function, the only residual signs now being a slight impairment of sensation and increased reflex activity in the lower limbs.

*Case 3.*—Male, age 34. Admitted to Wingfield Orthopaedic Hospital on Dec. 14, 1922.

**HISTORY.**—Pain in back and along costal margin for eighteen months. Kyphos over D.9 and 10. Wassermann, negative.

**X RAY.**—Destruction of bodies of D.9 and 10.

April 20, 1923.—*Graft.* Single tibial graft applied, long and broad, on left side.

Sept. 7, 1926.—*Re-graft.* Re-grafted from D.7 to 11 on right side, owing to disease being shown above original graft.

Dec. 17.—Spinal support applied.

March 29, 1927.—Discharged home.

Sept. 12.—Re-admitted, complaining of pain in abdomen and across back since May 1.

Dec. 8.—*Drags legs* as he walks. Right leg weak. Sensation: ? a little dull in right and left legs. Reflexes: knee-jerks + +; ankle-jerks + +; clonus (ankle) +; plantar, left extensor, right ? extensor (all others right and left).

Dec. 22.—Some difficulty in beginning micturition.

April 29, 1928.—Back on frame.

May 7.—Voluntary power: practically nil: can dorsiflex toes slightly. Spasm: +. Reflexes: knee-jerks + +; clonus (ankle) +; plantar, extensor (all right and left); abdominals + + above umbilicus, — below. Incontinence: difficulty in starting better since on frame.

May 10.—*Laminectomy.* Spines of D.9, 10, and 11 exposed. Graft most solidly fused to spines, cut away with difficulty with motor-saw, and preserved for re-insertion. Laminae of D.10 removed. No pulsation of dura. Probe passed downward freely, but not upward. Laminae of D.9 removed. Probe passed again upward tapped or ruptured a ? extradural cyst, and liberated a small quantity of turbid fluid. Flow did not continue, therefore not cerebrospinal fluid. Pulsation immediately started. A little more bone then removed in upward direction, and the old graft replaced, bridging gap. Operation followed by retention of urine and paralysis of both lower limbs of flaccid type, with partial loss of sensation in right leg and complete in left leg.

May 21.—Voluntary power: right and left legs, nil. Spasm: right and left legs, nil. Pinprick sensation: right leg, impaired; left leg, absent. Incontinence: rectal, complete; bladder, drained by catheter tied in and changed twice weekly. Reflexes: knee-jerks +; ankle-jerks +; clonus —; plantar, flexor (all right and left); abdominals upper abdomen +, lower abdomen —.

May 31.—*Return of spasticity.* Voluntary power: right and left legs, nil. Spasm: right and left legs +. Sensation: as on May 21. Incontinence: as on May 21. Reflexes: knee-jerks +; ankle-jerks + +; plantar extensor (all right and left); abdominals as on May 21.

June 30.—Automatic action of bladder established. No voluntary control.

Aug. 4.—Both lower limbs definitely spastic with increased reflexes; extensor,

plantar. Moving big toes and constant spasms. Complete anæsthesia and paralysis in left leg. Impaired sensation and slight power to move foot and toes in right leg. Bladder and rectum: automatic. No voluntary control.

Nov. 18.—Able to raise right leg off frame. No other change.

Jan. 22, 1929.—Discharged home at own request. During last four months the patient had curious periodical attacks of pyrexia lasting six to seven days, the temperature rising to a peak of  $103^{\circ}$  or so, and accompanied by severe headache and sweating, but no rigors. No cystitis was present. No important change in neurological state occurred. No recovery whatever of voluntary power or sensation in the left leg. Was able to raise the right leg and move the foot and toes, and sensation in this limb became practically normal. Both legs remained spastic, with spasms and increased reflexes and upward-moving big toes. Bladder and rectum: automatic, with no voluntary control.

**Comment.**—This is a case of late paralysis, onset five years after original admission. In addition the previous double grafting made laminectomy very difficult. This man died eighteen months later in coma.

*Case 4.*—Male, age 21.

**HISTORY.**—Pain and stiffness in back for six weeks, then fifteen days previously sudden onset of acute pain in back followed a few hours later by numbness and paralysis of legs. He was then admitted to the Radcliffe Infirmary, Oxford, and transferred to Wingfield Orthopædic Hospital on Oct. 29, 1923.

Voluntary power: right and left legs, nil. Spasms: right and left legs, nil. Sensation: complete loss below line posterior inferior spine, anterior superior spine, middle Poupert's ligament, symphysis pubis, right and left. No zone of hyperæsthesia. Reflexes: knee-jerks —; ankle-jerks —; clonus, nil; plantar, nil (all right and left); abdominals, upper +, lower —. Incontinence of bladder and rectum.

**Summary.**—Complete flaccid paralysis of both lower limbs, with loss of sensation and incontinence.

Oct. 30, 1923.—*Laminectomy and graft.* Laminæ of D.10, 11, and 12 removed. Pulsation free above, none in lower quarter of exposed area. After full exposure flattened area opposite D.11 filled out, but pulsation never seemed as full as at higher level. There was a prominence of the theca opposite D.11. Aneurysm needle passed in front of theca at this level failed to find debris. Double lateral tibial graft from D.8 to upper lumbar, bridging laminectomy gap.

April 28, 1924.—Nephrectomy (right) for pyelonephritis, with removal of ureter containing stone.

April 15, 1925.—Voluntary power: left leg greatly improved, right leg not so good. Spasms: left ++ (after effort), right ++ with very slight provocation. Sensation: no tactile sensation below either knee. Incontinence: complete.



FIG. 123.—*Case 4.* Skiagram showing bellows-like collapse of the vertebral body, with upper and lower discs little affected.

Aug. 31.—Walking with stick and one person's help. Has to wear urinal.

Oct. 30.—X rays show the collapsed vertebra with the intervertebral space above normal and below not obliterated—grafts in position (*Figs. 123, 124*).

Dec. 16, 1927.—Still incontinent and spastic.

Feb. 29, 1928.—Walking fairly well.

Feb. 19, 1930.—Walks with the help of two sticks quite well, though with stiff spastic type of gait. Voluntary power: practically normal in right and left legs;

dorsiflexion of feet incomplete owing to spasm of calf flexors. Spasm: right and left legs + (after exertion). Sensation: diminished though present below same line as before operation (right and left). Incontinence: voluntary control of bladder and rectum still not complete. Reflexes: knee-jerks + + +; ankle-jerks + + +; plantar, extensor; clonus (ankle and patella) +; abdominals + + (all right and left).



FIG. 124.—Case 4. Skiagram after laminectomy and grafts.

**Comment.**—Apparently the 11th dorsal vertebral body collapsed suddenly, and, shutting up like a pair of bellows, shot its semi-fluid contents backwards, causing so sudden a compression as to produce a traumatic flaccid paralysis. The radiogram (*Fig. 123*) shows the upper and lower discs of D.11 in contact. At the laminectomy it was clear that the theca was still compressed between the prominence in front and the laminae.

Laminectomy and graft was followed by considerable improvement, the paraplegia changing to the spastic type, and voluntary power returning sufficiently to permit of walking with a stick; but the patient has never recovered sufficient activity to resume working life. He still walks with a stick with a stiff spastic gait, is slightly incontinent of urine and faeces, and is liable to occasional spasms.

**Case 5.**—Male, age ? 35. Admitted to Wingsfield Orthopaedic Hospital on Oct. 17, 1923.

**HISTORY.**—Old tuberculous hip, and had three months' sanatorium treatment in 1917 for hæmoptysis; no tubercle bacilli found in sputum. Pain in back in March, 1923, followed by development of kyphos. Kyphos D.8 to 12.

**X RAY.**—Shows destruction of bodies of D.9 and 10, with collapse and lateral displacement.

Motor power: very spastic. Spasms: + + (right more than left leg). Sensation: right leg impaired. Incontinence: bladder distension, impairment of rectal sphincter control. Reflexes: knee-jerks + + (right more than left); clonus (ankle and patella) + (right more than left).

Nov. 22, 1923.—*Laminectomy and graft.* Three spines opposite kyphos, together with two above and two below, exposed. Laminae of three vertebrae removed. Dura

pulsated freely in upper part. No pulsation below middle of exposed area. The vertebral canal was much narrowed and distorted by double lateral curve so that the theca was confined and had been compressed over a distance of 2 to 3 in. After removal of laminae the compressed area filled out, but pulsation did not return to the middle and lower portion. Ridges of bony opening were smoothed and two tibial grafts inserted, bridging the laminectomy gap from two spines above to two below.

July 7, 1924.—Spasms much less. Improving greatly.

Oct. 1.—Off frame. Spinal support applied.

April 15, 1925.—Great improvement. Is wearing back splint at night to prevent contracture. Balance still poor. Legs still very spastic. Plantar response, flexor. Almost complete absence of sensation below knees. Walks with two sticks.

Feb. 1, 1926.—Re-admitted for pain over graft area. X-ray appearances suggested fractures of the grafts.

Feb. 8.—*Operation.* Grafted area exposed and inspected. No solution of continuity in grafts, which appeared firm and sound. Fusiform thickening of bone in centre of area possibly due to old fracture. Wound closed without further interference.

March 3.—Has resumed back support. No pain in back.

Oct. 30, 1927.—Admitted to Abingdon Sanatorium for some return of adduction spasm in legs. X rays showed solution of continuity of graft opposite point of maximum tension, but without any separation. Much granulation tissue round spine. Costotransversectomy considered.

July 9, 1929.—No pain in back. Less spasm in legs. Looking well.

Feb. 24, 1930.—Walks fairly well, but with dragging spastic gait. Voluntary power: practically normal. Spasms: + occasionally. Reflexes: knee-jerks ++; ankle-jerks ++; clonus +; plantar extensor (all right and left). Incontinence: still some weakness of bladder control.

**Comment.**—In this case also the onset was gradual and was well marked, though not complete, at the end of six months, the symptoms and signs here involving one limb more severely than the other. The laminectomy and graft was successful in relieving the paraplegia, but in sixteen months' time the patient was walking with two sticks and still spastic. He is able to get about with sticks and no pain, and has only minor discomfort from occasional muscular spasm and a slight persistent incontinence of urine. This and the persistent spasticity of his lower limbs debar him from leading an active working life.

**Case 6.**—Female, age 33. Admitted to Wingfield Orthopaedic Hospital on July 10, 1924.

**HISTORY.**—Previous history of tuberculous glands and pleurisy. Six months' history of pain and weakness in back. Tender over small kyphos in mid-dorsal region.



FIG. 125.—Case 6. Skingram showing large tense abscess in site of previous costotransversectomy.

X RAY.—Caries of body of D.8, angular deformity, and abscess shadow.

Central nervous system: legs spastic: knee-jerks + +; plantar, flexor. Signs of paraplegia increased. Spasms and tingling of legs became troublesome.

Oct. 28, 1924.—*Costotransversectomy* 9th right rib. Large abscess evacuated.

Oct. 31.—Now no spasms.

Nov. 11.—*Albee graft*.

Aug. 2, 1925.—Discharged.

Nov. 23.—Re-admitted after a fall leading to fracture of graft.

Nov. 24.—X rays show shadow of large tense abscess (*Fig. 125*).

Dec. 15.—*Repair of graft*.

Linear transverse fracture found about middle of graft. New 4 by  $\frac{1}{4}$  in. graft inserted into bed in old graft. This was made by a long vertical motor-saw cut to the left of the centre of the old graft so that with the main part of the graft left undisturbed the left side of it could be mobilized outwards.

*Re-opening of costotransversectomy*. About an ounce of pus and three small sequestra with bone sand removed. Toilet of wound with ether. Wound closed.

Dec. 22.—Turned. Sutures removed. Wounds well healed.

Jan. 23, 1926.—Turned. Wound corresponding to costotransversectomy broken down at area size of sixpence about its middle. General condition good.

Feb. 19.—Turned. Clear fluid, one or two drops only, from sinus, otherwise satisfactory. General condition very good.

March 16.—Off frame. Spinal support satisfactory.

April 5.—X rays show disappearance of the large abscess shadow (*Fig. 126*).

April 30.—Discharged. Still small sinus.



FIG. 126.—Case 6. Skiagram showing disappearance of the abscess after reopening of the costotransversectomy.

May 14.—Seen at home. Doing very well.

June 8, 1928.—Sinus healed. General condition good. To continue support.

Sept. 14.—General condition good. Leaving off support with no ill effects.

March 14, 1930.—Very well. Leading normal active life. Voluntary power: normal. Spasm: nil. Sensation: normal. Reflexes: knee-jerks + +; ankle-jerks +; plantar, not obtained; clonus —; abdominals + (all right and left). Incontinence: nil.

**Comment.**—In this case also there was gradual onset of paraplegic symptoms which never became complete. Costotransversectomy led to evacuation of a large abscess and resulted in disappearance of the pain and muscular spasms. A tibial graft was subsequently inserted, which fractured a year later as the result of a fall and necessitated a further operation for repair of the graft. At the same time the prevertebral abscess was again evacuated. Result: paraplegia cured.

**Case 7.**—Female, age 56. Admitted to Wingfield Orthopædic Hospital on Aug. 30, 1926, from Royal Berkshire Hospital.

**HISTORY.**—Ten weeks' pain in back. Gradual loss of power in legs now complete.

**X RAY.**—Much destruction of D.10. Spine: kyphosis involving D.9, 10, 11.

General condition extremely poor. Œdema of legs. Flaccid paralysis. Right knee-jerk, just obtainable. Foot-drop.

Later.—Knee-jerk +; ankle-jerk +; ankle clonus +; Babinski present (all right and left). Feet œdematous. No impairment of sensation.

May 12, 1927.—*Laminectomy and graft.* Theca not pulsating, pressed on backwards by sharp edge of bone in front. Collapsed a few hours after operation. Death.

**Comment.**—Gradual onset of paralysis becoming complete, at first flaccid, later becoming spastic in type. Laminectomy and graft was done, but the patient collapsed and died a few hours after operation. This patient was 56, very feeble, with œdematous legs, and operation was undertaken as a forlorn hope and only as the patient was going downhill with distress and great discomfort.

**Case 8.**—Male, age 33. Admitted to Wingfield Orthopædic Hospital on April 16, 1929.

**HISTORY.**—Pensioned from Army in June, 1928, with tuberculosis of the spine. Continued to get pain in back at intervals and was treated in University College Hospital and at Oswestry. Leather support fitted at latter.

In March, 1929, had 'influenza', and on getting about again noticed *weakness and numbness of legs, and difficulty in walking.*

**X RAY.**—Shows abscess formation at side of extensive caries involving D.10, 11, 12, and L.1.

April 16, 1929.—Voluntary power: gait spastic. Spasms: very slight and occasional. Sensation: normal. Reflexes: abdominals +; knee-jerks + + +; ankle-jerks +; plantar, extensor; clonus, nil (all right and left). Incontinence: nil.

May 21.—*Costotransversectomy.* Left side opposite centre of diseased area. Tuberculous granulomatous tissue and bone sand removed. Wound sutured.

June 21.—*Graft.* Three autogenous tibial grafts used and laid into bed prepared as follows: Two angled ones on right side slightly overlapping at the convexity of the kyphotic curve; one straight one on left side.

Sept. 13.—Off frame. Spinal support applied.

Oct. 13.—Began walking.

Nov. 21.—Last seen as out-patient. Voluntary power: no loss. Spasms: nil. Sensation: normal. Reflexes: abdominals +; knee-jerks + +; ankle-jerks +; plantar, not obtained; clonus, nil (all right and left). Incontinence: nil.

**Comment.**—This is a case of paraplegia appearing late, and, according to Madame Sorrel, of the incurable type. The paraplegic signs were motor only. They cleared up completely after costotransversectomy followed by grafting one month later. The man is leading an active life.

**Case 9.**—Male, age 6. Admitted to Wingfield Orthopædic Hospital on Dec. 20, 1928.

**HISTORY.**—Admitted to Radcliffe Infirmary on Dec. 8. History of ? tuberculous glands removed from neck a year previously. Weakness of legs and incontinence.

**X RAY.**—Shows collapse of D.6 and 7, with abscess shadows.

Dec. 20, 1928.—No deformity. Slight paraplegia. Some urinary incontinence. X rays show disease of D.6 and 7 (*Fig. 127*).

June 19, 1929.—Voluntary power: nil. Spasm: +. Sensation: normal. Reflexes: knee-jerks + +; clonus (ankle) + +, patella, occasional; abdominals—;

plantar, extensor (all right and left). Incontinence: automatic action of bladder and rectum with no voluntary control.



FIG. 127.—Case 9. Skiagram showing small abscess shadow.

July 22, 1929.—*Laminectomy and graft.* Laminæ of D.6 and 7 removed. Dura bulged backward, but probe passed easily up and down and laterally. No pus or granulatous tissue seen. Two grafts cut from left tibia and placed bridging laminectomy gap from D.4 to D.9, inclusive.

Feb. 7, 1930.—Paraplegia persists unchanged. The abscess shadow suggested tenseness, so costotransversectomy was decided upon.

Feb. 10.—*Costotransversectomy.* Grafted area inspected first through old incision. Grafts firm and solid, so not disturbed. Wound closed. Through separate incision vertebral end of 7th rib on left side resected. This was sufficient to liberate tuberculous pus and granulatous tissue without removal of corresponding

transverse process. As much débris as possible removed with spoon, and wound sutured with rubber tube drainage for twenty-four hours only.

Feb. 20.—Wound healed. There has been less incontinence since the operation. Neurological state in other respects unchanged.

July 17.—There is now commencing recovery of voluntary

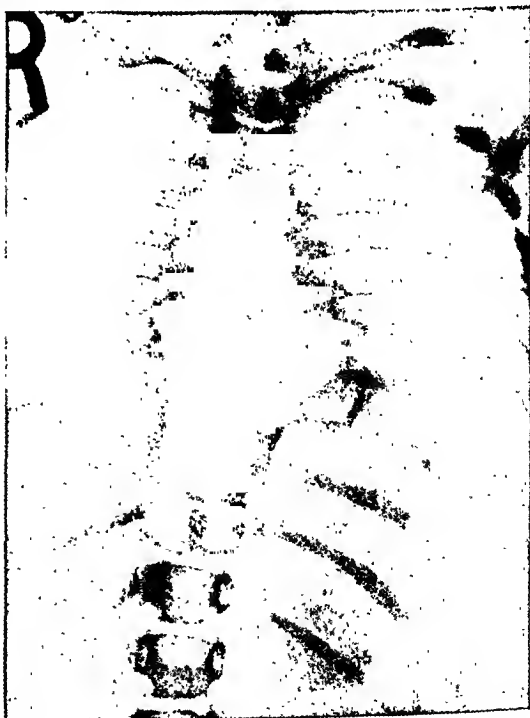
FIG. 128.—Case 9. Skiagram taken eight months after a large abscess had been emptied by costotransversectomy. Further accumulation of débris is shown.

power in the muscles of the thigh on both sides.

Oct. 10.—X rays show further increase of abscess (*Fig. 128*).

Later.—Voluntary power, sphincters, and sensation returned. Still some partial incontinence.

**Comment.**—A case of paraplegia of early onset, and in a



child, which increased during treatment and showed no sign of improvement after six months' immobilization. Laminectomy and costotransversectomy have led to a delayed but complete motor recovery. There is still incontinence. Presumably earlier operation would have had quicker and better results.

Further, the radiogram dated Oct. 10, 1930 (*Fig. 128*), shows a large abscess shadow. There has been a further accumulation of debris, indicating that it may be advisable to empty the abscess again through the costotransversectomy wound. Clearly general treatment and immobilization were not enough. In such a case one must interfere to relieve the cord from pressure, and be prepared to do so again if the signs and symptoms (always including radiograms) indicate the need.

*Case 10.*—Female, age 26. Admitted to Wingfield Orthopædic Hospital on Aug. 29, 1929.

*HISTORY.*—Confined in May, 1929. Since then has had pain in left thigh. Recently has noticed a lump in her back and has had difficulty in walking.



*FIG. 129.*—*Case 10.* Skiagram showing large tense abscess.



*FIG. 130.*—*Case 10.* Skiagram taken after laminectomy, grafting, and (a week later) costotransversectomy. The grafts and disappearance of the abscess are shown.

*X RAY.*—Disease D.11 and 12. Angular kyphos with lateral collapse and large abscess shadow (*Fig. 129*). Early paraplegia.

Voluntary power: both legs impaired. Ankle clonus present.

During the next six weeks the paraplegia deepened. Voluntary power: both legs, nil. Spasms: ++ (right more than left). Reflexes: knee-jerks and ankle-jerks normal (right and left); clonus, left +, right +++; plantar, extensor.



Sensation: anæsthesia to light touch over both heels, and impaired sensation on dorsum of right foot. Inability to distinguish pinprick over corresponding area. Trophic condition round feet: ankles very œdematous, and pressure-sores over anæsthetic area. Sphincters: normal.

Oct. 15, 1929.—*Laminectomy and graft*.

Oct. 22.—*Costotransversectomy*, 11th L., thus opening large abscess and evacuating much caseous material and sequestra. Sufficiently good exposure without removing transverse process.

Feb. 8, 1930.—X rays show disappearance of abscess shadow (*Fig. 130*).

Feb. 22.—Wound soundly healed; graft firm; no paraplegia.

Voluntary power: still slight weakness in right anterior tibial group, otherwise restored. Spasm: nil. Reflexes: knee- and ankle-jerks, normal; clonus, —; plantar, indeterminate. Sensation: no sores: no anæsthesia or hyperæsthesia.

March 21.—All muscles have now recovered and are functioning well, with the exception of extensor proprius hallucis.

May 4.—Back satisfactory; walking well. Some recovery of extensor proprius hallucis.

**Comment.**—Rather gradual onset, becoming complete in about two months. Laminectomy and graft was followed in a week's time by costotransversectomy in this case. The result has been almost complete recovery from paraplegia as early as four months after operation.

**Case 11.**—Male, age 34. Admitted to Wingfield Orthopædic Hospital on Feb. 1, 1930.

**HISTORY.**—Three to four months before admission noticed weakness of both legs and dragging of right foot. Unable to walk by end of December, 1929, and then first noticed difficulty with micturition followed later by incontinence. Incontinence of fæces about a month before admission. Complete loss of power in legs and spasms since December.

**X RAY.**—Disease involving D.3, 4, and 5. Abscess shadow on left side.

Feb. 1, 1930.—Definite kyphos at level of D.3. Voluntary power: nil. Spasms: +. Reflexes: knee-jerks +; ankle-jerks +; plantar, extensor (moving big toe); clonus —; abdominals — (all right and left). Sensation: impaired, but not lost to pinprick below line at level of 6th costal cartilage. Incontinence: automatic action of bladder and rectum, with no voluntary control.

Feb. 10.—*Laminectomy and graft*. Laminae of D.3, 4, 5 removed. Dura bulged backward and did not pulsate. Probe passed easily upwards and downwards. More bone nibbled away on left side, and the probe passed round, when a small quantity of caseous debris appeared and was removed. Pulsation was not observed to return. Two strong tibial grafts inserted on each side, bridging laminectomy gap from two spines above to two below.

Feb. 20.—Sensation is now normal except for a narrow band of anæsthesia below line at level of 7th costal cartilage. No return of voluntary power or sphincter control. No change in reflexes.

June 27.—Can definitely move voluntarily the outer toes on his right foot, and something in the outer part of the thigh. The effort initiates violent spasms. Spasms: severe at times.

Sensation: patient finds it very difficult to define his sensory reaction at all clearly. Undoubtedly he can appreciate pinprick everywhere over his lower extremities, though in patches he describes it as 'dull', and in places—especially the soles of the feet—as 'very sharp'. He is not consistent in these localizations, and in fact the reactions do seem to vary. There is a pretty constant line of hyperæsthesia about an inch above the umbilicus.

Incontinence: knows definitely when bladder or rectum is full, and has regained a measure of control over sphincter, though this varies from day to day.

Reflexes: abdominals —; knee-jerks ++; ankle-jerks ++ (right and left); clonus (ankle), right —, left +; plantar, right flexor, left extensor. Examination always starts severe spasm.

July 11.—Commencing recovery in muscles of thighs and legs.

Oct. 31.—Further improvement in voluntary power. Spasms still present. Sphincters still only partially under control.

**Comment.**—Gradual onset of paraplegia, taking three to four months to become complete. Laminectomy and graft has resulted in restoration of sensation to practically normal, and there is commencing return of voluntary power in the lower limbs and of control of bladder and rectum.

*Case 12.*—Girl, age 15½ years.

**HISTORY.**—In November, 1922, girdle pains associated with gradual loss of weight. In September, 1923, dorsal kyphos discovered. Treated in supine position till in June, 1926, she was pronounced cured and allowed up in celluloid jacket. In October, 1926, there was X-ray evidence of further abscess formation. She was sent to Leysin under Dr. Rollier. In February, 1927, she lost all power and sensation in her legs. After about a fortnight of complete paraplegia there is said to have been a gradual improvement, but in January, 1930, Rollier reported that the paraplegia had regressed.

Sept. 6, 1930.—Admitted to Wingfield Orthopædic Hospital. General mid-dorsal kyphosis. Complete motor, sensory, and sphincter loss below D.7. Spasms present.

Sept. 12.—*Laminectomy, bone-graft, costotransversectomy*—finding the theca was compressed by an encapsuled protrusion of caseous debris which had pushed its way backwards in the left side of the theca.

**Comment.**—The following points are noteworthy :—

1. Early girdle pains, ten months before a diagnosis of spinal caries.
2. After two years and nine months on her back the patient was allowed up, but within three months it was evident that the disease was still active. She was put on her back again, but in February, 1927—four years and three months after her first symptoms—complete paraplegia supervened.
3. This paralysis had persisted with temporary improvement for a further three years and seven months before any attempt at decompression.
4. It is a paraplegia of late onset, but an operable cause of pressure was found.
5. An operation so late in the course of paraplegia has relatively little chance of success.

### TECHNIQUE OF THE COMBINED LAMINECTOMY AND GRAFT OPERATION.

The patient is anaesthetized on his frame, or in cases of sharp angular kyphos on his plaster bed; then his turning-case is put on, strapped into place, and he is turned (*Figs. 131–133*). The anaesthetist arranges the flannel bandages between the anaesthetic bars so as to support the patient's forehead. It is very important for the anaesthetist at this stage to make sure that there is no edge of the turning-case pressing on the patient's neck or axilla. In this operation, amongst others in which a good deal of the body is covered only with sterile towels, we use double towels containing a layer of gamgee tissue between them in order to keep the patient warm.

The incision is slightly to the left side so as to keep the scar away from the spinous processes, and in order that the motor-saw may be used unhampered by retractors on the surgeon's side. The skin and subcutaneous tissue



FIG. 131.—Child on frame. Turning-case being applied.

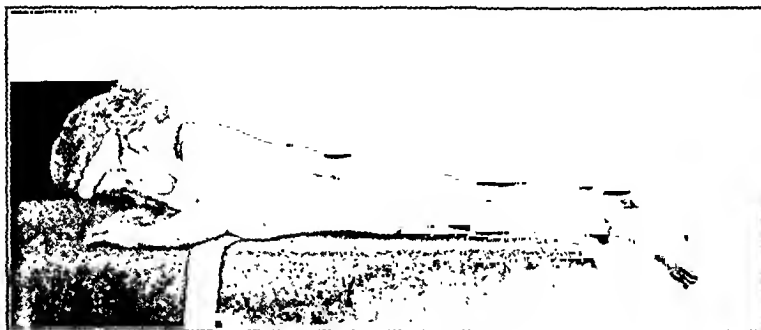


FIG. 132.—Child lying on turning-case as for operation.

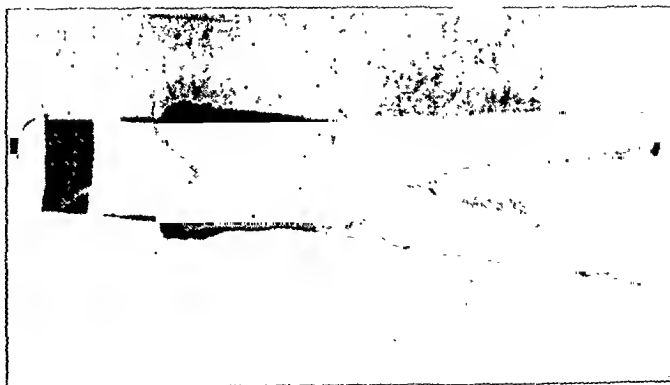


FIG. 133.—Turning-case fitted with anæsthetic extension.

are lifted from the deep fascia and reflected away from the surgeon beyond the line of spinous processes; the skin edges are closely and smoothly covered with fine towels.

Then an incision is made on to the apex of each spinous process and carried by a dip of the point of the knife through the interspinous ligaments over an area covering the three spines of the laminectomy area, two spines above, and two below. As the line of processes is often deranged by caries, and as it is best to make the incision run exactly over the apex of each process, it is helpful to define each process as the incision reaches it by gripping it between the points of a pair of dissecting forceps held in the left hand. The motor-saw is then taken and two incisions are made into each spinous process forwards and slightly outwards, to right and to left. These saw cuts are clearly shown on *Fig. 134*. They start in the knife cut and separate a thick flake of bone from the spinous process on each side. When each process has been dealt with in this way the saw is laid aside and an osteotome,  $1\frac{1}{2}$  in. wide, is used to complete the separation of the lateral flakes of bone from the central part of each spinous process, and then to carry these flakes outwards, together with the periosteum of the bases of the spines and laminae. I use an osteotome very broad so that it cannot possibly get between the laminae, and sharp so that it can be used gently without disturbing the carious centra. Each time



*Fig. 134.*—Showing motor-saw cuts into a spinous process.



*Fig. 135.*—State of affairs after reflection of osteoperiosteal flaps in the area of the laminectomy.

the osteotome travels outwards the space between it and the spinous process is packed with gauze, so that by the time the upper end is reached there is a thick packing all the way up that side. The other side is then treated in the same way (*Fig. 135*). It should be said that the laminae are only fully exposed where they are to be removed. In the case of the two spines above and below, the displacement of the flake of

bone and periosteum does not go beyond the base of the spinous process. The laminectomy is then done. It is essential to make the laminectomy opening of full width and long enough to relieve all the pressure present or at all likely to occur. *Case 1* is an instance of a laminectomy opening that was not long enough, though now I realize that the recurrent paraplegia could and should have been avoided by a costotransversectomy done before, or a week after, the laminectomy. I do not think I have ever had to remove

more than three laminæ for Pott's paraplegia. I do not open the dura, but 'seek' gently round it and sometimes evacuate a mass of débris from the side or the front of the theca.

Next comes the graft. The length and shape of the grafts required are recorded by bending a probe, and two 'Albee' grafts about  $\frac{3}{8}$  in. wide are cut

from the tibia. These can generally be straight. If so, they are put in with the periosteal surface deep—that is, with the smooth limiting membrane turned towards the theca. If the spine is straight or only moderately kyphotic, this gives plenty of clearance at the site of the lesion, because the grafts are carried fairly high up on the spinous processes above and below. If, on the other hand, there is much angular curvature, one of two methods must be used:—

1. *Shaped grafts*, to fit the angulation. This means using the

broadest part of the tibia, and not sparing the crest.

2. *Flexible grafts* (when a graft of sufficient length and angulation is unobtainable), prepared either by the earlier method of multiple transverse cuts about  $\frac{1}{10}$  in. apart on the medullary side of an Albee graft, and for this a very fast running saw is advisable; or by a technique in which one makes a number of parallel longitudinal cuts into the tibia  $\frac{1}{10}$  in. or less apart; after a transverse cut at each end six or more very thin and fairly flexible Albee grafts can be lifted from the tibia. Three or four of these are used on each side like laminated springs flexed to fit the angulation and firmly fixed to the spinous processes above and below. (I use a number of separate leaves. Albee, when lecturing at the Orthopædic Section of the Royal Society of Medicine in April, 1930, showed a lantern slide illustrating a graft in which a number of longitudinal incisions had been made but in which both ends were solid. In this case the range of flexion of the graft must be very small.)

When the grafts are firmly in place, and one has made sure that there

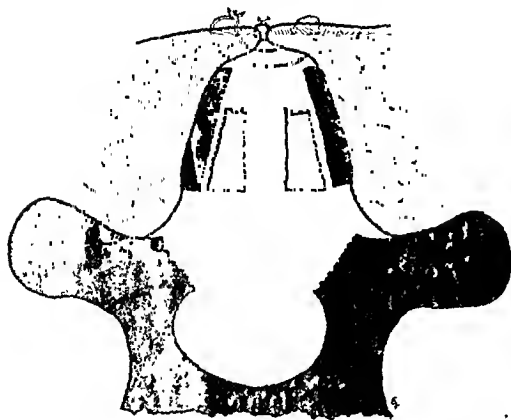


FIG. 136.—Grafts bridging defect left by laminectomy, with their lateral osteogenetic hosts.



FIG. 137.—Showing grafts as supported on spines above and below area of laminectomy.

is plenty of space between their deep surface and the theca, the edges of the supraspinous ligament, carrying with them the lateral flakes of bone from each spinous process, are sewn together over the grafts (*Figs. 136-138*). Just previous to this, if there are one or more prominent spinous processes, they are nipped across with bone-cutting forceps and bent under the suture line. This makes for comfort and safeguards the skin from pressure. The operation is completed by suture of the skin of the back and the leg. Then dressings and carefully graded layers of wool are applied, the frame or plaster bed is laid in position, the straps are pulled up, and the patient is re-turned.

**After-treatment.**—The patient remains on his frame or plaster bed for three or four months. He is turned whenever necessary by the method described (the routine in *all* cases of spinal caries), and each time the utmost care must be devoted to the exact adjustment and soft smooth padding of spinal frame or plaster bed.

Finally I would emphasize three things:—

1. I believe that if laminectomy is to be employed for Pott's paraplegia it should always be supplemented by grafting.

2. Good technique of such grafting is vital, for the grafts have to bridge the laminectomy opening without absorption, and because the strain on grafts in Pott's disease is often great. Therefore the grafts must be strong and be given a vascular and osseous bed or there will be risk of their absorption or fracture. It is for this purpose that I separate from each spinous process lateral flakes of bone with their periosteum intact, and a good vascular supply from the erector spinæ. At the end of the operation these lie with their raw surfaces in contact with the grafts; thus they provide an almost continuous series of vascular ossific nodes ready to vitalize and recolonize the grafts.

3. The continuous immobilization of the whole spine before, during, and after operation.

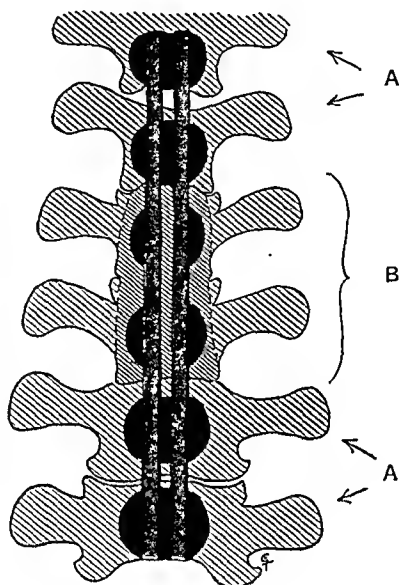


FIG. 138.—Diagrammatic coronal section of spinous processes and graft, with vertebrae and area of laminectomy indicated. A, Grafts well supported on spines; B, Area of laminectomy.

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**SIALOGRAPHY: ITS TECHNIQUE AND APPLICATIONS.**

By REGINALD T. PAYNE,

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SIALOGRAPHY is the radiographic demonstration of the ducts of the salivary glands by means of the injection of substances opaque to X rays. The method is of necessity one of strictly limited applicability, yet it may be claimed that its use has added to our pathological, diagnostic, and therapeutic knowledge of diseases of the salivary glands. In practice, anatomical considerations usually limit the investigation to a study of the ducts of the parotid gland, and although it has also been applied to the submaxillary gland, it is difficult to believe it can often be of use here. As an opaque medium, lipiodol has been found to be the most satisfactory, for owing both to its viscosity and its non-irritating nature it is less stimulating than the opaque bromides and iodides, which tend to be too rapidly ejected.

Although in the past sialography had often been carried out in the cadaver, Bársony,<sup>1</sup> writing in 1925, was the first to publish a case report where the method had been employed. Using a solution of 20 per cent potassium iodide he was able to demonstrate an enormous degree of dilatation of one parotid duct. This solution, however, gave rise to a considerable amount of pain. Of later workers, both Carlsten<sup>2</sup> (1926) and Keith<sup>3</sup> (1928), using lipiodol, have each recorded a case. The only record in the literature of the method as applied to the submaxillary duct is a scanty communication of Wiskovsky's<sup>4</sup> (1927).

**Indications.**—Briefly, sialography will demonstrate abnormalities, dilations, and obstructions of the larger and the smaller parotid ducts. It may, therefore, give evidence of a positive or negative value. For example, in the case of parotid calculi situated in the intraglandular part of the duct, radiography is often uncertain, and the employment of sialography may aid in exact localization. In the group of cases which fall under the heading of recurrent subacute parotitis of non-calculous origin—a condition which is usually a recurrent sialodochitis—the method may demonstrate certain well-defined lesions of the smaller ducts. Fistula cases, though rare in civil surgery, often present problems of a diagnostic and surgical nature which can at times be solved by sialography. Finally, the method may be employed for its negative value in such cases as true Mikulicz disease.

Further, it may be pointed out that whilst catheterization of the parotid ducts has an established diagnostic value, it is possible that the technique of sialography may possess a therapeutic value in the introduction of medicated substances into the salivary ducts direct.

**Technique.**—Sialography is not a difficult process, but is one requiring care and gentleness. From the point of view of the patient it is usually

painless, and the use of a local anæsthetic has the objection of tending to dry up all salivary secretions, thus rendering the entry of the duet more difficult. Only in one case (*Case 2*) was the completion of the injection followed by a transient swelling of the gland with some slight discomfort. At the end of twenty-four hours or less all the lipiodol has disappeared from the parotid duets.

*Apparatus.*—In carrying out the actual injection, home-made apparatus consisting of a rubber fountain-pen bulb attached to a short length of glass tubing, one end of which is drawn out to a fine point about 1 mm. in diameter, is the most satisfactory. It is usually best to have a series of these of graduated sizes. The glass tubing may either be straight or bent at a slight angle as shown in *Fig. 139*. The apparatus is sterilized either by boiling or by immersion in spirit before use. Round-end needles of No. 17 gauge and short lengths of fine ureteric catheters have both been tried, but the former is rather liable to damage the delicate structures of the parotid duet, and the latter is somewhat difficult to introduce. Further, it has been found much easier to steady in the duet the very light apparatus described, than to employ either of the other methods, which necessitate the attachment of a comparatively heavy Record syringe.

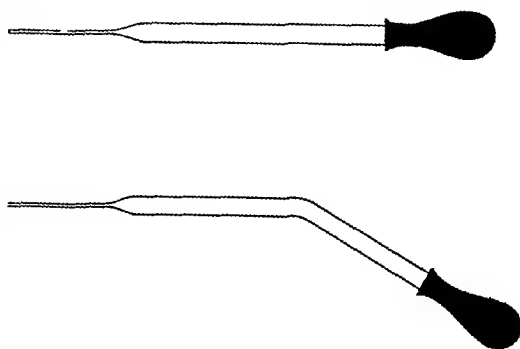


FIG. 139.—Type of syringe used for sialography.

*The Patient.*—Before beginning the process the patient washes out the mouth with water or a mild antiseptic. The orifice of the duct is then sought in the region of the second upper molar tooth, and gentle massage of the parotid will usually render it patent. If necessary, one or two of the graduated cannulas may be passed into it to dilate it slightly. In cases where the duet orifice cannot be identified, or where it is impossible to pass even the finest cannula, the patient is given a piece of lemon to suck. After this has been done there should be no difficulty in localizing the orifice.

*The Injection.*—The patient thus prepared lies on his back on the X-ray table with his head turned towards the sound side. The apparatus is filled with lipiodol, and the cannula introduced into the duet for about half an inch. Once in position, the apparatus should be held extremely lightly in the hand. The bulb is then squeezed gently for about half a minute to force the lipiodol into the duets. Screening is then carried out, and if the injection has been satisfactory, the main duct and the larger branches can be identified. If this is the case, the sialogram is taken. The injection process is then repeated, to be followed by a further screening and picture. As a rule  $\frac{1}{2}$  to 1 c.c. of lipiodol is all that is required. The development of pain over the parotid region or swelling of the gland indicates that no more lipiodol should be injected. Should these symptoms develop, they can be quickly relieved by massage carried out to empty the gland or duet.



## CASE REPORTS.

*Case 1.*—Glandular fistula (right parotid).

**HISTORY.**—E. M. C., a housemaid, age 45, attended St. Bartholomew's Hospital on March 6, 1930, complaining of a profuse discharge of watery fluid from below the right ear during meals. In 1927 she had been operated on for two abscesses in the right parotid region, one being just in front of the ear, and the other over the lower part of the mastoid process. The incisions healed up in the course of a few days. From the history it was impossible to be certain whether these lesions were due to suppurative lymphadenitis, or whether they were the result of suppurative parotitis. In January, 1929, she started to get a discharge from the lower part of the mastoid scar, which had gradually increased in amount. The discharge was always of a watery nature, and only occurred during meals, but at these times it was so profuse as to necessitate the patient's wearing a towel round the neck. Mumps had occurred during childhood.

**PHYSICAL SIGNS.**—The posterior scar was situated over the lower part of the mastoid process. At its lower end there was a pin-point opening from which saliva poured profusely when the patient was given a lemon to suck. The right parotid duct was normal and secreted freely, and no abnormality could be made out in the parotid gland itself.



FIG. 140.—*Case 1.* Sialogram. The opacity over the upper and posterior part of the second cervical spine corresponds to the opening of the fistula.

**INVESTIGATIONS.**—Ordinary skiagrams of the right parotid region failed to show any evidence of calculi. Sialography was carried out on March 20 to determine whether or not there was any communication between the fistulous track and any parotid duct of appreciable size. The sialogram (*Fig. 140*) shows the main duct of the gland, the larger branches, and the ductules, all well filled with lipiodol. The site of the fistula is marked by the opacity at the upper and posterior part of the second cervical spine, and there is no communication between this and the parotid ducts.

**TREATMENT.**—On May 12 the fistula was treated without anaesthesia by means of the electrocautery. The discharge of saliva ceased in about a week, and there has been no recurrence since then.

**Comment.**—In the usual classification of parotid fistulae into gland and duct types, the distinction is one based entirely on topographical anatomy. Further, this distinction is one which profoundly affects the surgical principles applicable to two types of fistula. The more exact knowledge, therefore, gained by sialography in showing the relationship between a fistula and the parotid ducts has a direct bearing on the question of treatment, especially where a so-called glandular type of fistula communicates with a comparatively large duct.

*Case 2.*—Recurrent subacute parotitis (right.)

**HISTORY.**—R. P., female, age 23, attended St. Bartholomew's Hospital on Oct. 16, 1930, complaining of recurrent attacks of swelling in the right parotid

region. The condition first started in 1927, the swelling always appearing during a meal, and once it was present lasting from one to seven days. The longest period of freedom from an attack had been six months, but during the past twelve months the attacks had been getting much more frequent. When the swelling was present it was painful, but not severely so, though at times it had been bad enough to make the patient stop eating, and had prevented opening of the mouth more than half way. The last attack started on Oct. 6, and the swelling increased a little with each meal, to diminish somewhat between meals. It finally cleared up on Oct. 10. There had at no time been any discharge into the mouth, though at times during the past year there had been a bad taste. At the age of 13 the patient had mumps.

**PHYSICAL SIGNS.**—No visible swelling of the right parotid region was present. On palpation there was a rather ill-defined swelling about an inch in diameter with its upper limit three-quarters of an inch below the zygoma and at the level of the external meatus. The swelling was situated over the masseter, and its forward limit corresponded to the anterior limit of the muscle. Clinically most of the swelling appeared to be rather above the level of the normal parotid duct. The orifice of the duct was normal, as were the orifices of all the other salivary glands.

**INVESTIGATIONS.**—Catheterization of the right parotid duct was carried out after the patient had been given a lemon to suck. The saliva which was obtained contained several elongated shreds of highly tenacious mucus. Examination of these shreds and also of the more fluid part of the saliva, by means of a Gram film, showed that pus cells were present from one to three per field. (The presence of pus cells in a catheter specimen of saliva is always definite evidence of an inflammatory lesion of the gland or ducts.) Skiagrams of the right parotid region showed no calculi. On Oct. 17 sialography was carried out without difficulty. Towards the end of the examination the patient complained of slight discomfort over the parotid region and there was at the same time a little fullness to be made out. The latter had entirely cleared up when the patient was seen again at the end of twenty-four hours. The sialogram, reproduced in *Fig. 141*, shows good filling of the parotid duct and its branches. In the anterior part of the gland there are in addition minute spherical dilatations present towards the terminations of the ductules—suggesting a 'bronchiectatic' condition. A further interesting feature is that the area where these dilatations are present corresponds to the position of the palpable swelling of the parotid.



FIG. 141.—Case 2. Sialogram showing the minute spherical dilatations towards the terminations of the smaller ducts in the anterior part of the gland.

**Comment.**—Recurrent subacute parotitis when not associated with the presence of calculi is invariably due to a mild degree of infection of the parotid ducts. The secretion in such cases is excessively mucoid in type, and at times plugs of mucus and debris corresponding to the main duct or its larger branches may be obtained by massage of the gland or by catheterization. Normally, parotid secretion is almost free from mucus, but traces are derived from glands situated in the larger ducts. In inflammatory

conditions this mucoid secretion is markedly increased, and together with pus cells and epithelial cells forms plugs which actually obstruct the ducts, and thus dam back the normal glandular secretion. In the present case the recurrent duct obstruction was undoubtedly the factor bringing about the dilatation of the smaller ducts in the anterior part of the gland.

Treatment in such cases should be directed towards the clearing up of all possible sources of buccal infection, and in addition, as a prophylactic measure, the patient should be shown how to massage the parotid gland and its ducts, in order to clear them of tenacious secretion. This should then be carried out for about five minutes before and after every meal.

*Case 3.*—Parotid calculus (left).

**HISTORY.**—E. M., age 34, male, clicker, attended St. Bartholomew's Hospital on April 14, 1930, complaining of a swelling of the left parotid region and a discharge into the mouth. In 1927 the patient first began to get a swelling of this region which came up during meals and then slowly went away afterwards. On the first occasion these symptoms were present for about a fortnight, and ever since then at intervals of a few weeks there had been attacks of a similar nature. In August, 1929, the swelling of the left parotid became very large and painful, and at the same time an ulcer developed inside the mouth in the neighbourhood of the duct, from which large amounts of foul pus poured. After a week the parotid swelling receded. Two weeks later the patient was operated on at Northampton General Hospital, for a small residual swelling near the angle of the jaw. No calculus was found and the wound healed by first intention. Ever since that time there had been swelling of the gland with every meal and a continual discharge of pus into the mouth.

**PHYSICAL SIGNS.**—About one inch above and in front of the angle of the jaw on the left side there was a slight visible swelling. Palpation revealed a small swelling about  $\frac{3}{4}$  in.  $\times$   $\frac{1}{2}$  in., firm in consistency, slightly tender, and not fixed to adjacent tissues. The duct orifice was reddened and slightly ulcerated, and pressure over the parotid gave rise to a flow of mucopus.



FIG. 142.—*Case 3.* Skiagram showing a calculus in the posterior part of the left parotid region. Its position is marked with the white dot.

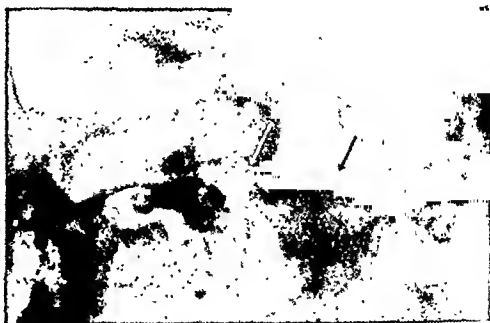


FIG. 143.—*Case 3.* Sialogram showing dilatation of the main parotid duct and absent filling of the branches. The position of the calculus corresponds to the ovoid dilatation at the posterior limit of the dilated duct. A certain amount of lipiodol is present in the floor of the mouth.

**INVESTIGATIONS.**—Examination of a catheter specimen of the pus showed the presence of pus cells in very large numbers, and on culture a pure growth of

*Staphylococcus aureus* was obtained. Ordinary skiagrams showed a small calculus in the posterior part of the gland (*Fig. 142*). On April 14 sialography was carried out without difficulty, and in *Fig. 143* the marked degree of dilatation of the main parotid duct is shown. At the extreme posterior limit of the duct there is a dense ovoid shadow which corresponds to the position of the calculus, and practically no lipiodol has got beyond this point. The other interesting feature is that none of the smaller ducts shows any filling at all. There is a certain amount of lipiodol present in the floor of the mouth.

**TREATMENT.**—On April 16 a small almost spherical calculus 5 mm. in diameter was excised from a markedly dilated duct. The wound healed by first intention, the discharge of pus into the mouth ceased in about a week, and the patient has remained free from symptoms since then.

**Comment.**—The points of interest in the investigation of this case are: (1) The degree of dilatation of the main parotid duct; and (2) The absent filling of all the branches of the duct. The explanation of the latter is probably that the preceding obstructive lesions followed by an acute inflammation had led to their obliteration.

#### Case 4.—Mikulicz's disease.

**HISTORY.**—A. E., male, age 48, attended St. Bartholomew's Hospital on March 12, 1929, complaining of swellings on both sides of the face and neck. About Christmas, 1928, the patient first noticed swellings in the parotid and submaxillary regions on both sides. Since they appeared they have tended gradually to increase in size, though from time to time the patient stated that they had varied somewhat. There had been no discomfort, and no disturbance of the general health.

**PHYSICAL SIGNS.**—Both parotid glands showed a visible enlargement of moderate degree, but did not overstep the normal anatomical limits. The overlying skin was healthy, the consistency firm, and no tenderness was present. The submaxillary glands were both enlarged to three times the normal size, but were otherwise normal. Neither the sublingual nor the lacrimal glands showed any enlargement.

**INVESTIGATIONS.**—Sialography of the right parotid gland was carried out on April 12 and showed no abnormality (*Fig. 144*). Ordinary skiagrams of the affected regions showed no abnormality. In addition, complete examination of the blood, including the Wassermann reaction, proved negative.

**Comment.**—In this case all the diseases known to give rise to Mikulicz's syndrome were excluded, and the condition was regarded as a true example of Mikulicz's disease proper of the non-familial type. The sialogram proved in addition that this disease is not associated with any gross lesion of the parotid ducts.



**FIG. 144.**—Case 4. Sialogram of right parotid in a patient with Mikulicz's disease proper.

#### SUMMARY.

1. The technique of sialography of the parotid, using lipiodol as a medium, is described, and it is shown to be a simple and a safe procedure.

2. By means of its application the normality or otherwise of the parotid duct and its branches can be demonstrated, and in pathological conditions affecting the gland the information so gained may render diagnosis more exact, and consequent treatment more rational.

3. Four cases are described where sialography has yielded useful information. In the first it was possible to demonstrate that a parotid fistula was strictly glandular in type; in the second a pathological dilatation of the smaller ducts was shown following recurrent subacute parotitis; in the third the markedly dilated condition of the whole of the parotid duct was shown in association with a calculus, together with absent filling of the smaller ducts; and in the fourth case the normality of the ducts was demonstrated in a case of Mikulicz's disease.

My thanks are due to Sir Holburt Waring, to Mr. L. Bathe Rawling, and to Mr. W. Girling Ball for allowing me to record these cases.

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## TWO CASES OF CARCINOMA OF THE SMALL INTESTINE.

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THE following cases are reported on account of the interesting contrast which they afford.

*Case 1.*—Male, age 54. The illness commenced with vomiting after the taking of rich food three months previously, and in the intervening period the patient had vomited every second day. When seen his appetite was poor, he had marked flatulence, and a feeling of fullness. His bowels had been kept regular by medicine. He had lost 3 or 4 lb. in weight.

The only positive finding on examination was slight fullness in the epigastric and umbilical regions. Gastric analysis showed nothing abnormal. X-ray examination revealed delay in emptying of the stomach, and marked dilatation of

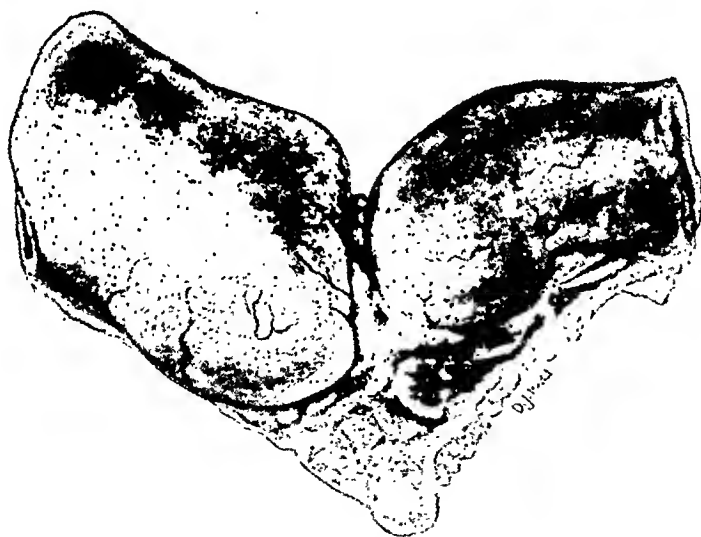


FIG. 145.—*Case 1.* Carcinoma of small intestine.

the small bowel with stasis. X-ray examination, then, pointed to obstruction in the ileum.

At operation the stomach and upper part of the small intestine were much distended; half-way down the small intestine there was a hard ring stricture causing almost complete stenosis (*Fig. 145*). In the mesentery adjoining the stricture there were two slightly enlarged glands. There was no evidence of metastases in other lymphatic glands, in the peritoneum, or in the liver. Resection was carried out, and the patient made a normal and uneventful recovery. Six months later he had gained 14 lb. in weight.

Examination of the bowel wall at the site of the stricture revealed an adenocarcinoma with considerable hyperplasia of fibrous tissue (*Fig. 146*). The lymph glands showed no metastatic deposits.



*FIG. 146.*—Case 1. Adenocarcinoma of small intestine, showing a more or less normal mucous membrano overlying the cancerous growth.

*Case 2.*—Male, age 57. The patient came complaining of a swelling above the right clavicle which he had noticed for the first time a month previously. He had been treated for neuritis, but this pain had largely disappeared.

Physical examination revealed a mass of enlarged glands in the supraclavicular region, and another mass in the right axilla. Hospital treatment was refused. A week later the man presented himself again, complaining of increasing pain and stiffness, and the glands were larger. Three days later he was admitted to hospital with several curious additions to his symptoms, a gradually increasing paralysis of the right side together with speech difficulty. Physical examination revealed a right-sided hemiplegia, flaccid in type, retention of urine, incontinence of faeces, and a complete motor aphasia. The glands in the supraclavicular region, however, were distinctly smaller. The patient became comatose and died.

At autopsy the jejunal wall, four feet below the duodenojejunal junction, showed externally a round flattened pigmented plaque, about an inch and a half in diameter. On opening the gut an ulcer with necrotic interior and rounded edges presented itself, but the lumen of the gut was unaltered in diameter (*Fig. 147*). The mesenteric glands near to the ulcer were enlarged and partly necrotic. The liver was large and riddled with growth, which was partly pigmented. The spleen was also entirely infiltrated with metastatic growth, the nodules being of varying size, and partly pigmented. Both lungs had scattered metastatic deposits, and the kidneys also showed a few nodules. The glands in the right supraclavicular triangle showed growth, and in the occipital lobe of the left cerebral hemisphere there was a large area of new growth, both necrotic and hæmorrhagic.

Sections of the edge of the primary ulcer (*Figs. 148, 149*) showed a more or less normal intact mucous membrane overlying a nodule of cells whose character was quite unlike that of glandular cells. These cells were oval or polyhedral, unequal in size, and of fairly uniform appearance. In places there was a suggestion of acini, but a suggestion only. The metastases showed a similar structure.

### COMMENT.

The two cases reported afford an interesting contrast in history, symptoms, microscopic histology, and malignancy. In the first case symptoms had shown themselves over a period of months. Vomiting had been more or less constantly present, appetite had been impaired, and the patient had lost



FIG. 147.—Case 2. Carcinoma of small intestine.

weight. The second patient had not suffered any ill health, and presented himself only shortly before his death because of the lumps in his neck. He did not call attention to his abdomen at all.

The first patient's cancer went on to stricture of the small gut with almost complete blocking. The second case, apart from the small localized ulcer, presented no alteration in the lumen of the gut.

The pathological histology of the first case was a typical very scirrhus adenocarcinoma. The histology of the second case suggests that the primary



growth may have arisen in a carcinoid of the jejunal mucous membrane at this point. As stated above, no glandular structure was observed, and there was a minimum of intercellular tissue.

The malignancy of the two cases also stands out in marked contrast.



FIG. 148.—*Case 2.* Carcinomatous change in carcinoid (?) More or less normal mucous membrane overlies the cancerous growth.

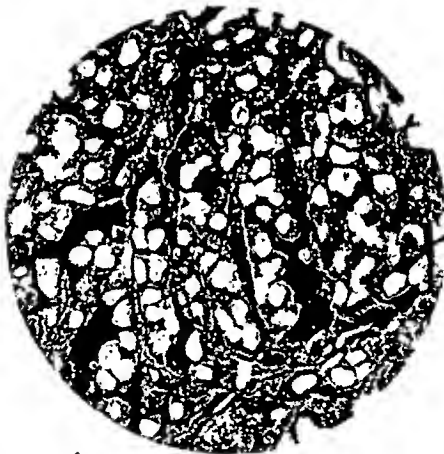


FIG. 149.—*Case 2.* High-power view of the cancer shown in Fig. 148.

In the first no metastases were discovered. The glands neighbouring the growth were not involved. In the second case, though the primary growth was so comparatively small, the metastases were widespread, glands, liver, spleen, lungs, kidney, and brain all being involved.

*SHORT NOTES OF  
RARE OR OBSCURE CASES*

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**A TUMOUR OF THE CAROTID BODY.**

By LAURENCE O'SHAUGHNESSY, OMDURMAN.

THE patient, a well-developed Sudanese of about 25 years, complained of a swelling in his neck (*Fig. 150*). He had been conscious of this for many years, but a rapid increase in size during the last few months had caused him to seek advice. Diagnosis seemed to lie between carotid tumour and parotid tumour, but the absence of pain and the position of the swelling favoured the former.

**OPERATION.**—The common carotid artery was ligatured with catgut, and by extending the incision the tumour was exposed. It was on the whole easily enucleated; only at its upper pole, which extended to the base of the skull, was any difficulty experienced. Separation from the carotid sheath was easily effected, although this was a point at which much difficulty had been anticipated. The wound was closed, and the patient made a good post-operative recovery. On the following day he was found to have a facial paralysis on the side of the operation.

**PATHOLOGICAL REPORT.**—I am indebted to Dr. E. S. Horgan for the following report:—

“The tumour was roughly oval in shape (15 cm. by 11 cm.), solid in consistency, with a well-marked fibrous capsule. On section it showed a series of irregular lobulated masses, gelatinous in appearance and of a greyish-white colour. In the centre of one was a small hæmorrhagic area.

“*Microscopical Examination.*—Low power (*Fig. 151*): The general structure consisted of solid masses of cells, which in some cases showed an alveolar or whorled structure; but such ‘whorls’ were ill-defined, and in many places the cells had no definite arrangement. The tumour had undergone extensive mucoid degeneration, and large areas consisted of myxomatous tissue with islands of tumour cells.

“High power (*Fig. 152*): The cells varied in shape and size, ranging from large polyhedral or spheroidal cells with well-marked granular cytoplasm



FIG. 150.—Tumour of the carotid body.

to smaller, more darkly staining spindle cells, some of which appeared definitely sarcomatous. There were numerous dilated capillaries present, but they appeared to bear no definite relation to the cell masses. Nor was it possible to trace any relation between their lining endothelial cells and the large spheroidal tumour cells.

"No pigment could be demonstrated in any part of the tumour.

"The long history of growth, the close relations with the carotid sheath, the well-marked encapsulation, and the general histological picture would suggest that this was a tumour arising from the carotid body.

"In the past various names like 'perithelioma' have been given to such tumours, but these



FIG. 151.—Low power. ( $\times 65$ .)

serve merely to confuse the issue. While the genesis and evolution of this neoplasm remain unknown it seems better to avoid labels."

**Comment.**—The mortality of this operation is high; Licini<sup>1</sup> gives it as 35 per cent. Beavan and McCarthy<sup>2</sup> consider that operation is not justifiable, and in their case employed radium with success. They state that only a small proportion of these tumours is malignant, and attribute the operative mortality largely to ligature of the carotid. My own experience of carotid ligature did not lead me to fear its effect, and, in the absence of radium, the only treatment for a rapidly growing tumour was extirpation.

I am indebted to the Director of the Sudan Medical Service for his permission to publish this note.

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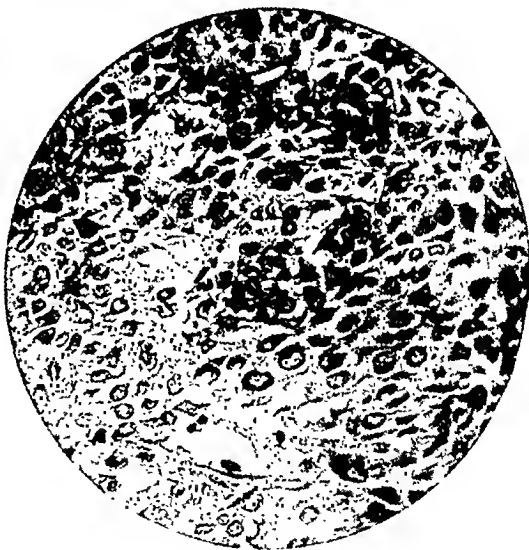


FIG. 152.—High power. ( $\times 230$ .)

## ANTERIOR DISLOCATION OF THE RADIUS AND ITS RECURRENCE.

By C. H. CORBETT,

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THE mechanism producing anterior dislocation of the radial head seems to be almost as great a riddle as that of anterior dislocation of both the radius and ulna at the elbow. Cohn<sup>1</sup> in 1922 found that not much could be added to the findings of Colson and Huguic's work of nearly a century ago, which were that anterior dislocation of both bones at the elbow was due to one of three causes: (1) Forced flexion of the forearm; (2) Upper arm fixation and turning the forearm round the forearm axis; and (3) Hyperextension of the forearm. In the mechanism of anterior dislocation of the radial head alone it seems probable that one or more of the above factors should exist. Cochrane<sup>2</sup> is of the opinion that the second and third factors are involved in anterior dislocation of the radial head, which is, he considers, due to a rotation of the forearm (radius) into pronation (i.e., 'rotation round the axis of the forearm'—No. 2 of Colson and Huguic's causes) plus hyperextension of the radial head through a fulcrum produced by an impinging of the radius upon the ulna immediately distal to the level of the radial tubercle. In the case set out below, the factor producing hyperextension was applied upon the distal end of the radius by the fall of the body thereupon, the arm being in pronation at the time. Added to and aiding these two factors, however, was the position of the forearm at right-angle flexion. Thus we see in the case in question the three factors involved were: (1) Flexion of the forearm; (2) Upper arm fixation and pronation of the forearm; and (3) Hyperextension of the radial head through a fulcrum produced by an impinging of the radius upon the ulna, probably immediately distal to the radial tubercle, and the force being applied by the weight of the body falling on the distal end of the radius. Thus we find that the general conclusions of Colson and Huguic nearly a century ago still hold.

Thomas's<sup>3</sup> view is very different, for in his discussion upon elbow fractures and dislocations, he says that the mechanism of anterior dislocation of the radial head is as follows: "If the elbow struck on an underlying stone or other such object, and this were of such size and shape and were so situated

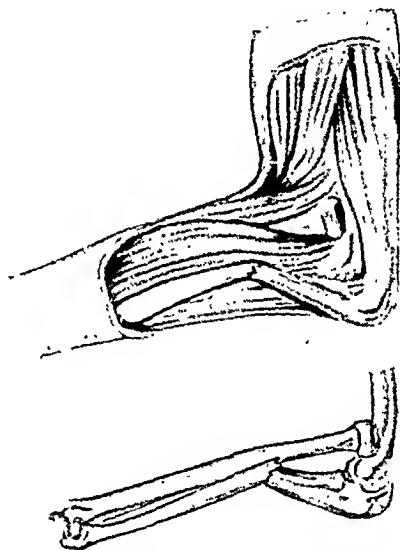


FIG. 153.—Fracture of the ulna in the upper third with dislocation of the capitellum (after Helferich), due to a force applied at the site of fracture and continued to produce dislocation.

that only the radial head received the impact upon it, we can easily imagine the force driving the radial head into anterior dislocation." It is difficult to imagine the conditions occurring which Thomas lays down for this dislocation—indeed, in the case under review the boy fell upon the smooth wooden floor, and though this type of dislocation is infrequent, it is common enough to suggest that other factors as suggested by Cochrane are usually at work. When one studies, however, the case of dislocation of the radius with fracture of the ulna, one sees a mechanism (*Fig. 153*) similar to what Thomas imagined might occur—namely, a fall on to the back of the flexed forearm which fractured the ulna at the junction of the middle and upper third and dislocated the radial head forwards. It must necessarily be an infrequent mode of dislocation, as the olecranon is such an efficient barrier and guard.

Scudder<sup>4</sup> does not publish his views upon the mechanism of this dislocation.

### CASE REPORT.

H. B., male, age 5. The patient was playing with some other children when he slipped upon a wooden floor and fell with his left arm bent under him, with a resultant dislocation of the radial head, his elbow being in flexion, the forearm in pronation, and the body falling over the distal end of the forearm. The dislocation was reduced by Dr. Kirk of Barton-on-Humber and kept in a sling for fourteen days.

The accident occurred on Oct. 19, 1927, and thereafter with trivial injuries there were subsequent redislocations on Jan. 24, 1928, Oct. 17, 1928, Nov. 15, 1929, and in January, 1930.

Broadly speaking, injury to the elbow region due to a comparatively slight accident such as a fall occurs in young subjects; even such an unusual accident as anterior dislocation of the forearm bones at the elbow occurs for the most part in subjects under 16 years of age, and in older people it is usually a very severe type of accident (a fall from a height, entanglement in a machinery belt, etc.) which causes the condition.

The boy in question was seen by me at the Surgical Out-patients' Department, Royal Infirmary, Hull, and was admitted to the wards on Feb. 13, 1930. It was judged that the orbicular ligament was beyond ordinary repair and that a plastic operation of some kind was indicated.

Campbell,<sup>5</sup> in 1929, discussing unreduced dislocations at the elbow, mentions the lack of particular satisfaction in obtaining fascia near the site of the dislocated radius, and ingeniously takes a strip of fascia lata, drills a hole in the ulna at the level of the radial neck, passes the fascial strip through the hole and around the neck, and sutures the orbicular ligament. It is to be doubted whether this is a facile procedure in the young subject and whether it is altogether necessary.

OPERATION (*Figs. 154-157*).—In the case under review it was considered that, given fascia stout enough for orbicular ligament repair and for strengthening of the stretched anterior capsule, the radial head should maintain its position, and it was hoped that the procedure of transference of part of the lacertus fibrosa (bicipital fascia) would meet the case (*Fig. 155*).

An incision (*Fig. 154*) was made parallel and external to the bicipital tendon and along the outer line of its fascia. The lateral cutaneous nerve was exposed and retracted, the cubital vein partially resected and ligated, the deep fascia

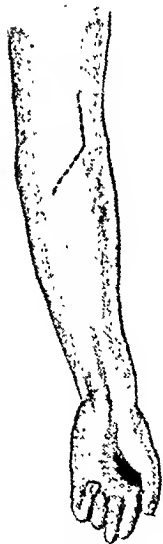


FIG. 154.—The line of incision.

divided, and the bicipital tendon with the lacertus fibrosa defined. The brachioradialis and extensor carpi radialis longior were brought into view and retracted, and the radial nerve thus exposed was retracted laterally with the above muscles. The supinator brevis was exposed and retracted downwards and outwards, and the capsule of the joint and the orbicular ligament were brought into the field. The external lateral ligament was defined in the lower three-quarters of its extent. The orbicular ligament was found torn and was fairly widely separated (Fig. 156).

At this stage a suitable strip of the lacertus fibrosa of about  $\frac{3}{4} \times \frac{1}{2}$  in. was taken and stitched with interrupted silk to the lateral ligament, capsule, and orbicular ligament as in Fig. 157.

**SUBSEQUENT HISTORY.**—The patient's elbow was maintained extended for a week, when light passive movement and massage were inaugurated, and rotation

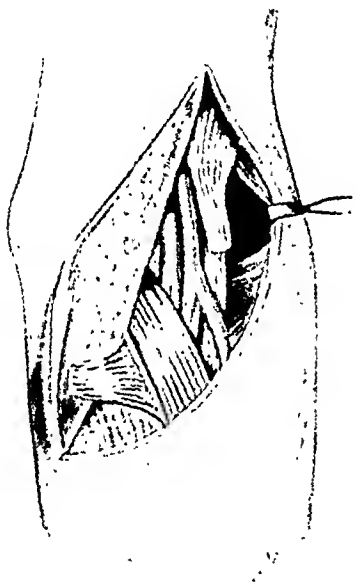


FIG. 155.—The approach to the part, with the lacertus fibrosa divided and attached distally.

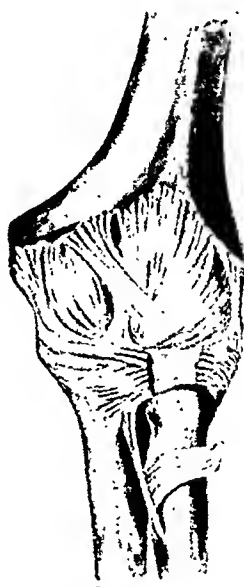


FIG. 156.—Diagram of separation of the orbicular ligament.



FIG. 157.—Method of stitching of fascial implant.

of the forearm was not permitted till a day or two before leaving hospital a fortnight later, when he was sent home to be under supervision and not allowed to use the arm for any but restricted movement for one month.

The operation was performed on Feb. 14, 1930, and now, April 23, 1931, he has a history of being the 'hope of his side' at cricket, and has had one or two severe falls upon his injured arm without untoward result.

The lesson of the operation is, that for children at any rate the lacertus fibrosa is an efficient transplant, and it seems probable that the adult lacertus fibrosa (bicipital fascia) is strong enough for a like repair.

#### REFERENCES.

- <sup>1</sup> COHN, I., *Surg. Gynecol. and Obst.*, 1922, xxxv, 776.
- <sup>2</sup> COCHRANE, *Fractures and Dislocations*, 2nd ed.
- <sup>3</sup> THOMAS, T. T., *Ann. of Surg.*, 1929, lxxxix, 108.
- <sup>4</sup> SCUDDER, *Treatment of Fractures*, 10th ed.
- <sup>5</sup> CAMPBELL, W. C., *Jour. Amer. Med. Assoc.*, 1929, xcii, 122.

## THE SUCCESSFUL REMOVAL OF AN OSTEOMA OF THE FRONTAL SINUS.

BY GEORGE ARMITAGE, LEEDS.

ROCKEFELLER FELLOW, 1929-30.

WITH reference to my article on osteoma of the frontal sinns that appeared in the April number of the BRITISH JOURNAL OF SURGERY, the following case may be of interest.

HISTORY.—Annie R., age 20, millhand, was admitted for a swelling in the frontal region which had been present for two years. When first noticed it was about the size of a marble, and in the middle of the forehead. It had increased gradually and painlessly until admission. There was a hard, nodular, fixed, and painless swelling in the mid-frontal region; it shelved off into the skull, but was more distinctly marked off above; below it reached to the supra-orbital ridges on both sides. The vertical measurement was  $2\frac{1}{2}$  in., the horizontal  $3\frac{1}{2}$  in. The skin over the tumour was freely movable. The patient suffered occasionally from slight headache, and thought the swelling might be due to a blow, but could not remember any.



FIG. 158.—Osteoma of the frontal sinus; anterior view. (*Specimen No. A.831, Pathological Museum, School of Medicine, the University of Leeds.*) ( $\times 1$ .)

OPERATION (April 29, 1903—the late Mr. Edward Ward).—A frontal flap was turned down and the tumour exposed. It was found to be exceedingly hard, and its surface was tuberculated. The bone around it was chiselled away, and then by means of a Wheelhouse's elevator it was levered out. It was found attached by a small pedicle, which passed down into the right infundibulum. During removal the dura mater was slightly torn, and was sutured. Iodoform gauze was packed down into both frontal sinuses.

The patient was discharged well on May 16. *Fig. 158* shows the specimen removed at operation.

## REVIEWS AND NOTICES OF BOOKS.

**Clio Medica Series.** A series of Primers in the History of Medicine. Editor: E. B. KRUMBHAAR, M.D. Fcap 8vo. 1930 and 1931. New York: Paul B. Hoeber Inc. \$1.50 per volume.

*The Beginnings: Egypt and Syria.* By WARREN R. DAWSON, F.R.S.E., Fellow of the Royal Society of Medicine, etc. Pp. 86 + x.

*Medicine in the British Isles.* By SIR D'AREY POWER, K.B.E., F.R.C.S., Hon. Librarian at the Royal College of Surgeons of England, etc. Pp. 84 + x. Illustrated.

*Anatomy.* By GEORGE W. CORNER, M.D., Professor of Anatomy in the University of Rochester. Pp. 82 + xvii. Illustrated.

*Internal Medicine.* By SIR HUMPHRY ROLLESTON, Bart., G.C.V.O., K.C.B., M.D., Hon. D.Sc., D.C.L., LL.D., Regius Professor of Physic in the University of Cambridge. Pp. 92 + x.

*Physiology.* By JOHN F. FULTON, M.D., Stirling Professor of Physiology, Yale University. Pp. 141 + xvi. Illustrated.

PROFESSOR KRUMBHAAR did well when, as editor, he dedicated this series of Primers to Clio, one of the Muses. It was the duty of the nine virgin goddesses to bring to the mind of the poet the subjects of his song; it was their pleasure to confer upon him the gifts of music and grace. Clio in particular was the Muse of History, and the Greeks showed her with a scroll in her hands or sitting beside an open chest of books. Like Janus, she looked before and after, for with her eight sisters she had the gift of prophecy, and told of things to come as well as of things past.

The five primers of the series which have already appeared show that the Muse was indeed a woman—*varium et mutabile semper*—for the history of medicine has many facets. Each facet has been polished by the skilled hands of Mr. Warren Dawson, Sir D'Arcy Power, Sir Humphry Rolleston, Professor G. W. Corner of the University of Rochester, and Professor John F. Fulton of Yale. The series therefore should be read as a whole, for it is only by doing so that the beauty and interest of the story can be adequately appreciated. Each volume is clearly printed, is of a size suitable for the pocket, and is provided with an index. Each, too, is introductory to the subject of which it treats, and a bibliography is provided for those who wish to learn more. The volumes are published in New York by Paul B. Hoeber at a price of one dollar and a half, which is the equivalent in England of 6s. 6d. There are more illustrations in the later volumes than in the earlier ones, and in future volumes the illustrations might well be even more numerous. It is clear that Professor Krumbhaar has chosen the writer of each volume of the series with great care, and it is interesting, therefore, to see how each author has treated his subject in relation to the whole body of the history of medicine.

The first volume deals with the 'Beginnings of Medicine in Egypt and Assyria'. It is written by Mr. Warren R. Dawson, who has already published a larger work on the same subject under the title *Magician and Leech*. In the early times, about which he writes, Medicine was still allied to Magic, though he traces the history to a time in Egypt when specialism had become well established, to a time told of by Herodotus when "some physicians are for the eyes, others for the head, others for the teeth, others for the parts about the belly and others for internal disorders." Cyrus in those days sent to Egypt for a physician, believing that the Egyptians had the highest reputation for their skill in the healing art—an opinion endorsed by Homer.

Sir D'Arcy Power has limited himself to medicine in the British Isles, and shows how medicine and surgery in England have developed along lines which differ entirely



from other countries. Until lately the State has been unconnected with doctors and their affairs, which had gradually developed in the case of surgery from a religious trade guild into a profession. Relapses occurred from time to time, and advances were made by a few upright men at irregular intervals, but it is only in recent years that it has been possible to unite surgery with its sister branch of inner medicine. The story is well told, and Sir D'Arcy has added chapters on Nursing, on the Medical Societies, and on Some Masters of Medicine, taking John Arderne, William Harvey, Thomas Sydenham, William Smellie, William and John Hunter, Edward Jenner, Dr. Bright, Thomas Addison, Dr. Hodgkin, Sir James Y. Simpson, Sir William Stokes, and Lord Lister as examples.

Dr. George W. Corner has written a thoroughly reliable account of anatomy couched in so light a vein that it is eminently suitable for those who have but little medical or scientific knowledge. He has taken a wide view of the term 'anatomy' and has not confined himself either to anthropotomy or to comparative anatomy, but deals with histology and embryology. He devotes some wise words to current trends in anatomy, and if he is true to the principles enunciated, the University of Rochester will have cause to congratulate itself on having secured his services as Professor of Anatomy.

Sir Humphry Rolleston devotes a volume to a survey of the history of internal medicine, by which he means the old-fashioned term 'physic', and not the whole of the healing art. Beginning with Jewish, Hindu, and Chinese medicine, he tells of Greek medicine and of the manner in which it is linked with modern medicine. He then considers the Renaissance, which witnessed the revival of anatomy, physiology, and the application of chemistry and physics to the problems of medicine; and lastly he gives a most excellent account of the school at Leyden, famous for Hermann Boerhaave, 'the Dutch Hippocrates'. A chapter is devoted to the advances made in all directions during the last century, of which the present generation are the fruitful heirs.

The last volume of the series to appear is devoted to physiology, and is written by Dr. John P. Fulton, who was until recently a Fellow of Magdalen College, Oxford. It is designed perhaps more for medical students than for the general public, but it is well illustrated, is well written, and is easy of comprehension by those who only have the elements of physiology. Dr. Fulton begins by showing the influence of Aristotle and Galen which gave physiology a better start than was obtained by anatomy. He then proceeds to consider physiology under the headings of the vascular, the respiratory, and the digestive systems, with a chapter on the physiology of the nineteenth century and an excellent account of the rise and development of the teaching laboratories. The epilogue on the trend of modern physiology is of especial value, and should be read and reflected upon by all who are interested in the subject of medical education and of the part in it played by the ancillary sciences.

In conclusion, the publishers are to be congratulated on the enterprise which has led them to launch the series, which is likely to become a success commercially. They announce twelve more volumes on the same lines as being in preparation, and promise other volumes in addition.

Sir D'Arcy Power: *Selected Writings, 1877-1930.* Medium Svo. Pp. 368 + x. Illustrated. 1931. Oxford: The Clarendon Press. 25s. net.

THIS volume, forming an anthology of his writings, was presented to Sir D'Arcy Power by a number of his friends to celebrate his seventy-fifth birthday, Nov. 11, 1930. It contains three surgical and thirteen historical papers out of more than six hundred articles and volumes, which have proceeded from his pen. We are sure that most readers of this fascinating volume will want to see more of the remaining five hundred and ninety. For besides his eminence as a surgeon there is no one who has made the history of medicine in England in the past so thoroughly his own as Sir D'Arcy, no one who can make Harvey, Hunter, or Arderne so live again before us, while he explains their hopes and work by details which he has gathered from a

thousand sources during fifty years of ceaseless study and writing. Nothing startles the casual reader more than his minute knowledge of very minor actors who may be mentioned, for instance, in Ward's *Diary*.

The pathos, too, of many of his pictures is very great. For instance, take his story of Hunter, that "fierce rude prophet", crippled and hindered in his life's work not only by his poor education and the imperfect science of the time, but still more by the terrible anginal and cerebral attacks, the result of the syphilitic inoculation which he foolishly tried on himself twenty-six years before his death.

One of the most pathetic stories in the English language is that of poor Pepys detailing month by month the torments he suffered in his work from bad eyestrain, probably hypermetropic astigmatism, and his final discourse with himself on the necessity of at once stopping his beloved, his incomparable, *Diary*. This was thirty-four years before his death. What a brilliant picture of the Revolution we have lost by this stoppage! In our author's hands the accounts of Pepys' constant breakdowns and his efforts to find some kind of glass to relieve him become a great tragedy the sadness of which is increased by the reflexion that, though scientific treatment was then unknown, relief might probably have been had at once if anything had led him to read through a slit while using his glasses.

If anyone thinks that medical history is void of tragic interest, let him read Sir D'Arcy's vision of the deaths of Wallace, Wat Tyler, and the martyrs at the gate of St. Bartholomew's, or even the story of poor Dr. Harvey in his sixtieth year, riding forty miles a day on a mission in Italy and then imprisoned in a filthy lazaretto and attacked by sciatica, as he details his woes in letters to Lord Feilding and implores his help for release.

In these selected papers we have an outline of the story of surgery in England from 1300 to 1850 with charming sketches of many of the actors. The author begins with his hero Arderne, and the *De Arte physicali* which he has since translated and edited. But we must go to the Early English Text Society for his delightful biography of Arderne and other translations of his works.

In discussing the various classes of mediæval medical men he touches on the difficult question as to what surgeons had the title of 'Magister', suggesting that, besides University graduates, all members of the guild of surgeons enjoyed it, but we find this very guild appointing four of their members a year to be Magistri or overseers. Still it is possible that the suggestion is correct, and when late on the absurd title 'Master' Doctor Harvey turns up, we have to confess almost anything seems possible.

Sir D'Arcy remarks on the unique use of the word 'furlong,' (p. 44), as a measure of time. We would suggest that it is like the 'time of a Paternoster' given in sundry receipts. Indeed, the Oxford Dictionary, quoting a like phrase from the *Miller's Tale*, says: "They sitten stille wel a furlong way", and in *Two Cookery Bookes* we read, "Let them stonde a furlong way or two." On p. 60 he quotes another archaic phrase, "a minute of an hour", i.e., 60 seconds.

Again, while the statement on p. 45 that hospitals had no separate medical staff before the sixteenth century may be true in a sense, it is clear that physicians were wardens in some cases—for example, Dr. Paseal, of Bonona, was Master at Preston in 1265, and Dr. Recouchez at Pontefract Hospital. Indeed, at York there was Ann Medica as a sister, and of course in monasteries there was always an official Infirmarius.

It is interesting to learn that the best mediæval surgeons were often itinerant, living with the patient till he was cured, and paid partly in cash and partly by an annuity. The description of the education of a surgeon under Vicary and his successors is excellent, but we wish that more emphasis had been put on the fact that much of the system existed earlier than 1540; thus the Royal Charter of 1462 confirms to the Company of Barbers and Surgeons the right of examining surgeons, and their Ordinances in 1529 enforce attendance on the weekly lecture on surgery for those on the surgical side.

In his account of Harvey, Sir D'Arcy suggests as a possible cause of Harvey's coming to St. Bartholomew's his friendship with Inigo Jones, who was born in the

Hospital precincts and was studying in Italy at the same time that Harvey was there. Whether that is so or not, young Harvey after his return married the daughter of Dr. Lancelot Brown, and in 1609 got a royal recommendation and was appointed Physician. His service there lasted some forty-three years, but in the last twelve he was often compelled to be absent either in personal attendance on the king, or when sent on a royal mission. The mission in 1636 to Vienna and Italy under the Earl of Arundel makes an extraordinary story, the details of which were first discovered by Sir D'Arcy. He shows us the embassy making its way through the horrors and devastation of the Thirty Years War, the crowds dying of starvation whom they had to relieve, and the freebooters whom they had to fight at every turn. At last they reached Vienna and Prague, and had audiences with the Emperor, but Harvey was sent on alone to Venice about some pictures for the king. It was on this four-hundred mile ride that he was imprisoned at Treviso, but he finally carried out his mission, and after a visit to Florence and Rome rejoined his party in safety. The whole forms a striking episode in Harvey's life and a good picture of travel at the time. It is curious to compare with it Nicholas Ferrar's journey over much the same ground twenty years earlier and his detention in a lazaretto.

The paper on the first part of John Ward's *Diary* is of very great interest as a picture of the lives and work of Willis, Lower, and other scientific physiologists just before the foundation of the Royal Society, an epoch more fruitful and important in English medicine than any other till the nineteenth century. The author shows that Ward actually records Joliffe's discovery of the lacteals a year or so before any one else published a claim.

The Bradshaw Lecture on cancer of the tongue was well worthy of inclusion in this volume. It brings forward so many facts which are apt to be overlooked, and attains to some curious results. The lecturer made a remarkable search in ancient and mediæval literature to find out in what degree the disease was prevalent in the past. To his surprise he could not find a certain instance of it before the case given by Alex. Read in 1635. Since then there has been a huge increase in male patients, while in women and animals it remains comparatively rare as before. After reviewing many statistics on this disease and on the co-existence of syphilis and dental caries, he concludes that a great predisposing cause of the increase has been old and inadequately treated syphilis and alcohol. The exciting cause is usually irritation from tobacco and in a lesser degree from dental caries, but none of these things will produce it apart from the presence of the unknown specific cause.

The history of the Royal College of Surgeons of England will appeal to everyone with its amusing accounts of the lectures and the fate of Hunter's Museum, Smollett's description of an examination, and its many stories of Abernethy, Blinck, Sheldon, and South.

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A Text-book of the Surgical Dyspepsias. By A. J. WALTON, M.S., M.B., B.Sc. (Lond), F.R.C.S., Surgeon, London Hospital; Late Hunterian Professor, Royal College of Surgeons. Second edition. Demy 8vo. Pp. 720 + viii, with 286 illustrations in the text and 2 plates. 1930. London: Edward Arnold & Co. 42s. net.

THE second edition of this book presents a clear account of the practice of the author and his views on those conditions which give rise to upper abdominal discomfort. As is to be expected, definite and almost dogmatic views are expressed in relation to the conditions discussed, and the author's reasons for holding them are given. We can see and follow his line of thought in every instance, although we may not always be in complete agreement with him. Special mention must be made of the chapter on the difficult subject of visceroptosis. The clinical picture is admirably drawn, the indications for operation are carefully considered, and the conclusion, with which most of us will agree, is reached that operations are, as a rule, to be avoided. It is perhaps unfortunate that there is no mention of operations on the abdominal sympathetic, but possibly it is not considered that their results are as yet sufficiently uniform for them to be included in a book of this kind.

In the chapter on gall-stones it is recommended that cases of acute cholecystitis should be operated on without delay. Certainly a well reasoned justification is

given, but we are inclined to think that the dangers of delay are rather exaggerated, and that the fact that the normal course for cases allowed to settle down is tranquil and not attended by complications is not sufficiently brought out. It may be sound theoretical reasoning to compare the gall-bladder with the appendix, but practical experience shows us that the two viscera do not behave in the same way when inflamed. Exploration of the common bile-duct from within is advocated in every operation on the biliary system. We do not agree with this, as there is no doubt that careful palpation from without, rolling the duct between the finger and thumb, will reveal not only a stone but even grit to an observer who has trained himself by always examining the duct in this way.

The chapters on the stomach and duodenum are admirable; they are lucid and complete, and it is only in details that we can offer any criticism. For example, in the description of the operation of gastro-enterostomy, it is stated, "the excess of mucosa is not removed as this step appears to increase the likelihood of a gastro-jejunal ulcer." That is a very important statement, and is contrary to the experience of many surgeons. To give it without any justification or explanation is not enough to make a surgeon who does not believe it even consider it. In discussing perforated ulcers it is said that the pain may be sufficiently severe to cause the patient to writhe about. We cannot accept this. The typical picture is one in which the patient lies absolutely still, in which the breathing is entirely thoracic, and in which it appears as if he is afraid that the slightest movement might cause an exacerbation of his suffering. We are very much in agreement with the opinion which is voiced that operation should be undertaken at the earliest possible moment. To wait for an improvement after the collapse which is seen in the first hour or two is to wait for something which will not occur. The condition of the patient and the prognosis become worse with each succeeding hour. In the treatment of a perforated ulcer it is said that the fluid which has escaped should be mopped out and that when drainage has been instituted the pelvic tube may be left in position for ten days. If it is recognized that this mopping out must only be done in the mildest of ways and that perhaps any solid matter which is seen may be removed, then it is harmless. To attempt to clean out the fluid present in anything like a methodical manner is to increase the shock of the operation by a procedure which is unjustifiable and unnecessary. The time the tube is left in position is a matter of opinion, but we cannot agree that by rotating it each day the danger of ulceration into the bowel can be avoided.

Finally, we cannot agree with the author in his treatment of the appendix during the course of an abdominal operation for some other condition. He states that he finds evidence of disease in only 4 per cent of cases and that he only removes the appendix in those cases. An appendicectomy does not appreciably prolong the time taken by the operation, and it is a safeguard for evermore against the development of an appendicitis. It has been truly said that the only disadvantage is that it derives some surgeon of a fee at a future date.

This is a book which represents very clearly the surgical view in cases of dyspepsia; it embodies the teaching and practice of the large majority of surgeons who are engaged in this kind of work in a way in which no other text-book with which we are familiar does. If it is a little individual in parts, that is only what we can expect from an author with the skill and experience of Mr. Walton. The publishers are to be congratulated on the clearness of the text and on the way in which they have reproduced the large number of really useful illustrations.

Diseases of the Tongue. By WALTER G. SPENCER, M.D., F.R.C.S., Consulting Surgeon, Westminster Hospital; and STANFORD CARR, F.R.C.S., Assistant Surgeon, Westminster Hospital. Being the third edition of *Baillière's Diseases of the Tongue*. Demy 8vo. Pp. 561 + xvi, with 123 illustrations in the text and 29 coloured plates. London: H. K. Lewis & Co. Ltd. 25s. net.

THE success of radiotherapy in buccal cancer has led to a revival of interest in diseases of the tongue, and for a monograph on such a subject there could

value of immediate stabilization operations for cases of compression fractures or other fractures of the spine without cord lesions is not mentioned.

When the author does leave his narrow plaster pathway to describe operative methods, we think that he is confused partly because he uses complicated devices. For example, the open operation for comminuted fractures of the humerus is difficult either to understand or believe in. Fractured olecranon has to be treated by an axial screw—which, of course, will take no hold on the loose cancellous tissue of the ulnar shaft—then the limb is to be put up in plaster for four weeks! If an open operation is properly done on the olecranon, no splint is required and the patient should be back at work in four weeks. The same kind of criticism applies to the operation for fractured patella, which has to be ‘plastered’ for five weeks.

We think it deplorable that a modern book on fractures should devote so much attention to adhesive traction appliances for leg fractures and so little to transfixion pins or the taut wire traction. We do not agree that it is never necessary to do an open operation for fractures of the tibia and fibula. As a clear exposition of an excellent method the book is good. As a general exposition of the treatment of fractures it is too limited to be of any real value.

**Cancer and Race: a Study of the Incidence of Cancer among Jews.** By MAURICE SORSBY, M.D., F.R.C.S.E., Hon. Assistant Surgeon, Ear, Nose, and Throat Department, London Jewish Hospital, etc. With a Preface by Lieut.-Col. F. E. FREMANTLE, M.A., M.D., M.Ch., F.R.C.P., F.R.C.S., D.P.H., M.P., Consulting County Medical Officer of Health for Hertfordshire. Conducted under the auspices of the Jewish Health Organization of Great Britain. Demy 8vo. Pp. 120 + xvi. 1931. London: John Bale, Sons & Danielsson, Ltd. 7s. 6d. net.

This is an interesting short study designed to throw light on the problem of the racial factor in the causation of malignant disease. For a long time there existed a vague and non-substantiated idea that Jews were immune, or at any rate less liable to cancer than non-Jews. This question has been settled once and for all by the writer. He shows that Jews as a race neither enjoy an immunity nor suffer from a predisposition to cancer, and that as communities they show vagaries towards a higher or lower incidence of cancer which are similar to the larger communities in which they reside.

The author, however, calls attention to the variation of the incidence of cancer of certain organs in Jews. He gives evidence for his belief that the incidence of cancer of the penis, the tongue, and buccal cavity is lower in Jews than in non-Jews, whereas the incidence of cancer of the intestinal tract is higher. He attributes the low incidence of cancer of the penis, which is shared by Mohammedans, to circumcision, and not to any racial immunity. The low rate of cancer of the tongue he believes is connected with the low rate of syphilis among Jews, and the low rate of cancer of the uterus to the beneficial rules of sexual hygiene which were imposed by the Mosaic Law. No explanation for the high incidence of intestinal cancer is given. The conclusion of the argument appears to be that, in the incidence of cancer, Jews do not show any specific racial characteristic.

**Le Cancer.** By G. ROUSSY. Second edition. 1929. Pp. 846, with 280 illustrations. 1929. Paris: Masson et Cie. Fr. 100.

This book forms the second volume of the great system of medicine published under the auspices of Roger, Vidal, and Teissier. The work represents one of the most complete discussions of our present knowledge of tumours available in any language. Like so many French books, it suffers from the grave defect of having no index, is printed on poor paper, and bound in a peculiarly fragile material. However, many of the pictures are inset on good art paper and cannot fail to be of great value. The book starts with a short introduction to the history of our knowledge of cancer; then a long chapter deals with those agents known to be capable of producing malignant tumours, and there is here an excellent account of the various forms of cancer associated with industrial processes.

The section on pathogenesis is short, and contains little more than a résumé of the various hypotheses that have been published everywhere. Almost 250 pages are devoted to the pathological anatomy and histology of malignant tumours, and here many excellent photomicrographs and drawings have been included. The English reader may find some difficulty in this section because of the terminology used in France. The custom of referring to almost all forms of carcinoma as epithelioma is confusing at first, but this is only a temporary difficulty. A particularly good part of this chapter is the description of tumours affecting the nervous system. About 150 pages are devoted to a discussion of the pathological physiology of the cancer process, and there probably exists no better description of the natural history of the disease.

Malignant tumours as they occur in the lower animals are briefly described, and there is an excellent description of the experimental production of cancer. A short but interesting section is that devoted to the tumours occurring in plants. The general symptomatology of malignant tumours is described in detail, as are various diagnostic methods. Only about 150 pages are devoted to the discussion of treatment. Individual surgical procedures are not described, but radiological therapy is discussed at some length.

The last seventy pages of the book are of great interest as giving statistical information of the mortality from cancer in most of the civilized countries. In the last few pages the author describes the steps being taken in different countries to enlighten the general public as to the nature and prevention of malignant tumours.

The whole book is a monument to the industry of Professor Roussy and his two collaborators, Dr. Roger Leroux and Dr. Maurice Wolf.

*Physiologie pathologique chirurgicale. Inflammations, Effets des Traumatismes, Réparation des Plaies, Greffes, Maladies des Os, des Articulations, des Vaisseaux et des Nerfs.* By R. LERICHE, Professeur de Clinique chirurgicale à la Faculté de Strasbourg; and A. POLICARD, Professeur d'Histologie à la Faculté de Lyon. Medium 8vo. Pp. 212. 1930. Paris: Masson et Cie. Fr. 26.

This little volume, in common with many another work associated with Leriche, will be found to be not only of interest to those who are trying to correlate recent advances in physiology with the problems of surgery, but also a thought-provoking stimulus to everyone who is attempting to understand the nature of disease as a whole. It is not merely a collection of new facts; it is more a criticism of commonly accepted explanations of certain phenomena in the light of new knowledge, and the statement of a fresh point of view. There is scarcely a page in the book that does not call for an exercise of the critical faculty, and time and again the reader is overcome either with the delight of seeing some well-known phenomenon in a truer light, or with a burning desire to argue with the author. It is the product of a fertile mind; problems with a vision unclouded by preconceived ideas, and is of value not only for the thoughts expressed therein, but for the ideas for further research which a critical reader may obtain from its study.

An attempt is made to elucidate the nature of the process of disease; and in the case of diseases of unknown cause it is hoped that by studying their effects and expressing the particular tissue reactions in terms of physiology as at present understood, the cause may eventually be discovered. As the authors state in their introduction, the volume is intended to encourage the clinician to attack his problems from the standpoint of the experimental pathologist, and the pathologist to study disease while function alone is affected and before the stage of structural change has been reached.

Seeing that function is given the most prominent place, it is natural that the importance of tissue nutrition should be stressed, and this gives Leriche the opportunity to bring forward his favourite subject of vasomotor control. His statement, "It may be said that our physical and psychic existence is nothing but a series of vasomotor reactions" may be regarded as the text and keynote of the whole work; yet one cannot help noting the remarkable success which he records following

operative procedures designed to interrupt the paths along which he believes the controlling impulses to travel.

In a work of such a character it is difficult to single out any chapters for special praise. Those dealing with inflammation and arthritis are excellent, but the most striking are the sections on bone—especially the mechanism underlying the absorption and deposition of bone in pathological states—and on the arteries and veins. The way in which changes in the blood-vessels are linked up with clinical phenomena, and more especially with sensations of pain, is most remarkable. And most astonishing of all are certain of the methods recommended for the relief of such pain—methods the value of which is vouched for by the authors after fifteen years of careful research.

This book is to be heartily commended, not because it is startling and unorthodox and entertaining, though it may be considered to possess all those attributes, but because it will be illuminating and give food for thought to all who are interested in advancing the science of surgery.

**Surgical Diagnosis.** By American authors. Edited by EVARTS AMMOSSE GRAHAM, A.B., M.D., Bixby Professor of Surgery, School of Medicine, Washington University, St. Louis, etc. Volume III. Large 8vo. Pp. 1043, with 446 illustrations. 1930. Philadelphia and London: W. B. Saunders Co. Per set—three volumes and index volume—£7 10s.

THE first two volumes of *Surgical Diagnosis* were reviewed in Vol. XVIII, No. 69, of this JOURNAL. Volume III is now presented, together with a separate Desk Index Volume, which is a general index to the three volumes. It deals with the surgery of the thorax, breast, liver, pancreas, rectum, genito-urinary organs, and nervous system. The remarks made previously concerning the first two apply equally to this volume. It is a useful work of reference for the general practitioner who wishes to steer his patient through difficult times.

**The Metabolism of Tumours.** Investigations from the Kaiser-Wilhelm Institute for Biology, Berlin-Dahlem. Edited by OTTO WARBURG, Kaiser-Wilhelm Institute for Biology, Berlin-Dahlem. Translated from the German edition, with accounts of additional recent researches, by FRANK DICKENS, M.A., Ph.D., whole-time worker for the Medical Research Council, Courtauld Institute of Biochemistry, Middlesex Hospital, London. Demy 8vo. Pp. 327 + xxix. Illustrated. 1930. London: Constable & Co. Ltd. 40s. net.

THE work of Warburg and his school on the respiratory processes in living cells, and especially in those of tumours, is too well known to need discussion. Much of this work is at present too theoretical to be of immediate interest to the practising surgeon, but to the research worker on cancer it is of very great interest and importance. This translation of the German edition of Warburg's collected papers, together with a number of recent additions, will be heartily welcomed by all workers concerned in cancer research.

**Guy's Hospital Reports.** Edited by ARTHUR F. HURST, M.D. Vol. LXXXI (Vol. XI Fourth Series), No. 1. January, 1931. Royal 8vo. Pp. 126. 1931. London: The Lancet Ltd. Annual subscription £2 2s. net; 12s. 6d. per issue.

THIS number contains a symposium on streptococcal infections, which is similar to a symposium on staphylococcal infections appearing in the number for April, 1930.

Dr. Ryle deals with the natural history, prognosis, and treatment of streptococcal fever in an article notable for its restraint and sound common sense. Perhaps the most valuable part of his article is the review of the various forms of treatment which have been keenly advocated by enthusiasts. Of serum therapy he says: "On the whole, we must confess that there seems very little evidence in favour of the efficacy of serum therapy. It is very generally employed, and had its effects been in any way dramatic there must by now have been a stronger consensus of opinion to support it." Again, his conclusions concerning intravenous medication

are worth quoting: "Merenrochrome and other chemical antiseptics by the intravenous route are to be avoided. There is no sound evidence, whether experimental or clinical, that they do good. Some of them have undoubted toxic effects. Their administration can be distressing to the patient and they are capable of causing disturbing symptoms."

G. F. Gibberd writes the article on streptococcal puerperal sepsis, and it is interesting to note that he too looks with disfavour on methods designed to kill the streptococci in the blood-stream. He discusses the place of blood transfusion in puerperal streptococcal infection, and defines it as follows: invaluable as a prophylactic measure after hæmorrhage, and as a therapeutic measure nearly always good, provided the reaction does not kill the patient.

Dr. Mollison describes the streptococcal infections of the throat. Mr. Layton describes hæmolytic streptococci in the mastoid, and Dr. Barber those of the skin, while Dr. Knott deals with the streptococci from the standpoint of the bacteriologist.

## BOOK NOTICES.

*[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.]*

**Beobachtungen am gebrochenen Knochen.** By Dr. C. BAUER (Köln a. Rh.). Würzburger Abhandlungen Bd. 26, H. 12. Super royal 8vo. Pp. 432-448, with 12 illustrations. 1931. Leipzig: Johann Ambrosius Barth. RM. 1.60.

**The Veterinary Journal.** Edited by FREDERICK HOBDAV, C.M.G., F.R.C.V.S., F.R.S.E., Hon. Veterinary Surgeon to H.M. the King. Special surgical number. Royal 8vo. Pp. 107-158. Illustrated. March, 1931. London: Baillière, Tindall & Cox. 2s. net.

**Recent Advances in Radiology.** By PETER KERLEY, M.B., B.Ch. (N.U.I.), D.M.R.E. (Camb.), Assistant Radiologist, Westminster Hospital. Demy 8vo. Pp. 324 + viii, with 120 illustrations. 1931. London: J. & A. Churchill. 12s. 6d. net.

**Crippled Children: their Treatment and Orthopedic Nursing.** By EARL D. McBRIDE, B.S., M.D., F.A.C.S., Instructor in Orthopedic Surgery, University of Oklahoma Medical School. Large 8vo. Pp. 280, with 159 illustrations. 1931. London: Henry Kimpton. 15s. net.

**Verlauf der wichtigsten Knochen- und Gelenkerkrankungen im Röntgenbilde. Eine anschauliche Prognostik.** By Privatdozent Dr. med. VICTOR HOFFMANN, Oberarzt der chirurgischen Universitätsklinik im Augusta-Hospital zu Köln. English and German text. Imperial 8vo. Pp. 264 + x, with 584 illustrations. 1931. Berlin: Julius Springer. Paper covers, RM. 66; bound, RM. 69.80.

**Die Bakteriologie der Wurmfortsatzentzündung und der appendikulären Peritonitis.** By W. LOHR (Magdeburg) and L. RASSELD (Altona). Royal 8vo. Pp. 95 + viii, with 46 illustrations. 1931. Leipzig: Georg Thieme. RM. 12.

**Röntgendiagnostik der Gallenblase.** By Priv.-Doz. Dr. F. EISLER and Dr. G. KOPSTEIN. Radiologische praktika Bd. XVII. Super royal 8vo. Pp. 153 + viii, with 151 illustrations. 1931. Leipzig: Georg Thieme. Bound, RM. 18.60.

**Operative Chirurgie der Knochenbrüche.** By Professor FRITZ KÖNIG (Würzburg). Vol. I. Operationen am frischen und verschleppten Knochenbruch. Royal 8vo. Pp. 194 + vi, with 200 illustrations. 1931. Berlin: Julius Springer. Paper covers, RM. 27; bound, RM. 29.80.

**Die Lebensvorgänge im normalen Knorpel und seine Wucherung bei Akromegalie.** By J. ERDMANN, A.O. Professor an der Universität Wien. Pathologie und Klinik in Einzeldarstellungen, Bd. III. Large 8vo. Pp. 160 + viii, with 31 illustrations. 1931. Berlin and Vienna: Julius Springer. Paper covers, RM. 18; bound, RM. 19.60.

**Collected Papers 1904-1929.** By EDWIN BEER, M.D. (New York). Large 8vo. Pp. 827 + xii, with 252 illustrations. 1931. New York: Paul B. Hoeber Inc. \$7.50.



# 170 THE BRITISH JOURNAL OF SURGERY

**Thomson and Miles' Manual of Surgery.** By ALEXANDER MILES, M.D., LL.D., F.R.C.S. (Ed.), Consulting Surgeon, Royal Infirmary, Edinburgh; and D. P. D. WILKIE, M.D., F.R.C.S. (Ed. and Eng.), Professor of Surgery, University of Edinburgh. Vol. I. General Surgery. Eighth edition. Crown 8vo. Pp. 574 + xvi, with 176 illustrations. 1931. London: Oxford University Press. 12s. 6d. net.

**Das Pollersche Verfahren zum Abformen an Lebenden und Toten sowie an Gegenständen.** By ALPHONS POLLER. Edited by E. B. POLLER and E. FERSCHER. With a Foreword by Prof. Dr. C. von ECONOMO (Vienna). Large 8vo. Pp. 216 + xii with 129 illustrations. 1931. Berlin and Vienna: Urban & Schwarzenberg. Paper covers, RM. 12; bound, RM. 14.

**Cancer: International Contributions to the Study of Cancer.** In honour of James Ewing. Edited by FRANK E. ADAM, M.D., F.A.C.S., Attending Surgeon to the Memorial Hospital, New York. Super royal 8vo. Pp. 484 + xx, with 168 illustrations. 1931. London: J. B. Lippincott Co. 45s. net.

**Surgery: its Principles and Practice. For Students and Practitioners.** By ASTLEY P. C. ASHURST, A.B., M.D., F.A.C.S., Professor of Clinical Surgery in the University of Pennsylvania. Fourth edition, thoroughly revised. Large 8vo. Pp. 1189 + xii, with 1063 illustrations and 15 coloured plates. 1931. London: Henry Kimpton. 45s. net.

**La Chirurgia della Innervazione periferica del Simpatico (Chirurgia del Dolore).** By Prof. IGNAZIO SCALONE (Milan). Large 8vo. Pp. 254 + xvi, with 91 illustrations. 1931. Milan: Ulrico Hoepli. L. 30.

**Abdominal Pain.** By JOHN MORLEY, Ch.M., F.R.C.S., Assistant Surgeon, Manchester Royal Infirmary, etc. With an Introduction by J. S. B. STODDARD, M.D., F.R.S., Professor of Anatomy, University of Manchester. Medium 8vo. Pp. 191 + xvi, with 22 illustrations. 1931. Edinburgh: E. & S. Livingstone. 10s. 6d. net.

**Practical Methods in the Diagnosis and Treatment of Venereal Diseases for Medical Practitioners and Students.** By DAVID LEES, D.S.O., M.A., M.B., D.P.H., F.R.C.S., M.R.C.P. (E.), Surgeon in Charge of Venereal Diseases, Royal Infirmary, Edinburgh, etc. With an Introduction by WM. ROBERTSON, M.D., F.R.C.P., D.P.H., Late Medical Officer of Health, Edinburgh. Crown 8vo. Pp. 634 + xx, with 87 illustrations. 1931. Edinburgh: E. & S. Livingstone. 15s. net.

**L'Ostéosynthèse des Os longs. Etude critique, biologique et pratique.** By C. E. CORNIOLEY (Geneva). Preface by Prof. CH. JULLIARD. Royal 8vo. Pp. 422 + xvi, with 102 illustrations in the text and 16 plates. 1931. Paris: G. Doin et Cie. Fr. 75.

**Injuries and Sport. A General Guide to the Practitioner.** By C. B. HEALD, C.B.E., M.A., M.D. (Cantab.), M.R.C.P. (Lond.), Physician, with charge of Electrotherapeutic Department, Royal Free Hospital, etc. Demy 8vo. Pp. 543 + xxiv, with 280 illustrations. 1931. London: Humphrey Milford. Oxford University Press. 25s. net.

**Clinical Observations on the Surgical Pathology of Bone.** By DAVID M. GREG, M.B., C.M., F.R.C.S.E., F.R.S.E., Conservator of the Museum of the Royal College of Surgeons of Edinburgh. Large 4to. Pp. 248 + xii, with 224 illustrations. 1931. Edinburgh: Oliver & Boyd. 30s. net.

**Surgery.** By S. K. KEN, L.R.C.S., L.R.C.P. (Edin.), L.R.F.P.S. (Glas.), late State Surgeon, Nepal. Vol. III. Surgery and Pathology of Growths. Demy 8vo. Pp. 318 + viii, with 56 plates. 1931. Calcutta and Burdwan: The Surgical Education Society Ltd. Rs. 7/8 per vol.

**Guy's Hospital Reports.** Edited by ARTHUR F. HURST, M.D. Vol. LXXXI (Vol. XI Fourth Series), No. 2. April, 1931. Royal 8vo. Pp. 127-252. Illustrated. London: The Lancet Ltd. Annual subscription, £2 2s. net; 12s. 6d. per issue.

**A Textbook of Surgery.** By JOHN HOMANS, M.D., Assistant Professor of Surgery. Compiled from Lectures and other writings of various members of the Surgical Department of the Harvard Medical School. Royal 8vo. Pp. 1195 + xii, with 513 illustrations. 1931. London: Ballière, Tindall & Cox. 40s. net.

**Röntgenuntersuchung und Strahlenbehandlung der Speiseröhre.** By Dr. JOSEF PALUGYAY (Vienna). Handbuch der Röntgenkunde, Vol. III. Royal 8vo. Pp. 392 + xiv, with 224 illustrations. 1931. Vienna: Julius Springer. Paper covers, RM. 56; bound, RM. 50.60.

# THE BRITISH JOURNAL OF SURGERY

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## *SOME BYGONE OPERATIONS IN SURGERY.*

BY SIR D'ARCY POWER, K.B.E., LONDON.

### VI. AMPUTATION: THE OPERATION ON NELSON IN 1797.

The history of amputation has still to be written. The operation must always have been necessary, but there does not seem to be any detailed account until Celsus describes it in the last chapter of the seventh book *De Medicina*, and then only in a few lines as a treatment for some cases of gangrene. He recommends what is now called the modified circular operation. The skin and muscles are to be divided and retracted, the bone being sawn through at a higher point. There is no mention of any method of arresting hæmorrhage or of bringing the edges of the wound together. Archigenes, who is mentioned by Juvenal, recognized that amputation was required in cases other than gangrene, but neither he nor Paulus Aegineta, who lived some five hundred years later, altered the time-honoured circular division of the limb, though they stopped the bleeding by applying very hot irons. Amputation, even in cases of gangrene, seems to have fallen into complete disuse amongst the Arabian surgeons, and it was not until the thirteenth and fourteenth centuries that it was revived by Henri de Mondeville and Gui de Chauliac, both of whom used the circular method.

It is certain that the circular method remained in exclusive use for very many years. John Woodall, writing in 1639, says:—

"The amputation once resolved upon and all things ready for the work, let the Surgeon with all his assistances and friends not forget before the beginning of the work heartily to call upon God for a blessing upon their endeavours, and let the patient the day before have notice given him that he also may take time to prepare himself with true resolution of soul and body to undergo the work, as being never performed without danger of death, which done, then let the Surgeon prepare himself also with his helpers, namely at the least five persons besides himself, as for example, one to sit behind the patient to hold him, a second for a holder, who by the surgeon must be instructed to stand fast before him and to bestride the limb to be amputated and to amplex the limb; and a third to hold and stay the lower end of the diseased member

to be taken off; a fourth to receive and bring back the sharp instruments; a fifth, to attend the Artist and deliver to him his needles and buttons, restrictive rollers, bolsters, bladder and so soon as possibly may be to stay with the palm of his hand the medicines applied to the end of the abscissed stump that being the duty of the fifth helper and the sixth is the Artist himself that dismembereth. Six and not fewer are the least for the work of taking off a leg or an arm if it be done in the whole or sound part or for taking off a member proceeding by a wound by Gun-shot, done in the lacerated not totally mortified part. But for the taking off a member in the mortified part three persons as assistants may serve, or two for a need, namely one to hold the upper part, the other the lower end. Let the surgeon have ready for instruments a fit amputating Cerra [saw], a Catlin and a good small incision knife, a good pair of strong scissors and three or four cauterising instruments. Let one of the assistants take the upper part of the member, holding it in both hands reasonably fast and steady. I mean the whole part thereof somewhat near unto the unsound part and let the other helper hold the other part. I mean the putrid part to be abscissed in his hand whilst the Surgeon himself first by circumcising divide the putrid flesh from the bone doing it somewhat near the quick part but not too near it, about an inch full from the quick part that with the Cerra he may come without fear to divide the bone or bones asunder where he is sure they are sphacelated not touching any quick part at all with his sharp instruments which he may observe the certainer to do, if with a Needle he enquire cautiously. Let him also divide betwixt the bones the parts there being lest by lacerating or tearing with the teeth of the Cerra he offend, which done let the Artist amputate the bones."

Amputation was still almost limited to cases of gangrene, for the means of arresting hæmorrhage were crude, difficult, and painful.

Forty years after Woodall had thus described the operation in general use, or perhaps sooner, C. Lowdham, a Devonshire surgeon of whom little is known, was practising an entirely new method, a flap operation. This was adopted by his fellow-countryman James Yonge, who published in 1679, "A new way of amputation and a speedier convenient method of curing stumps than that commonly practised, in which (are) divers other useful matters recommended to the military Chirurgeon." The communication took the form of a letter addressed to Mr. Thomas Hobbs dated from Plymouth, August 3, 1676, and it appeared as pages 108-120 of his pamphlet "*Currus Triumphalis, è Terebinthô, or an Account of the many admirable Vertues of Oleum Terebinthinæ.*" Thomas Hobbs, to whom the letter was written, was Lithotomist to St. Bartholomew's Hospital and Serjeant Surgeon (1687-97) to James II and William and Mary.

The manner of the new operation, says Yonge, was as follows, and the first hints came "from a very ingenious brother of ours Mr. C. Lowdham of Exeter. The Ligatures and Gripe being made after the common manner you are with your Catlin, or some long incision-knife, to raise (suppose it the Leg) a flap of the membranous flesh covering the muscles of the Calf, beginning below the place where you intend to make excision and raising it thitherward of length enough to cover the stump. Having so done turn it back under the hand of him that gripes; and, as soon as you have severed the member, bring this flap of cutaneous flesh over the stump and fasten it to the edges thereof

*Currus Triumphalis, è Terebinthô.*

OR AN

Account of the many admirable Vertues

OF

*Oleum Terebinthinæ.*

More particularly, of the good effects produced by its application to recent Wounds, especially with respect to the *Hemorrhagies* of the Veins, and Arteries, and the no less pernicious weepings of the Nerves, and Lymphaducts.

Wherein also, the common Methods, and Medicaments, used to restrain *Hemorrhagies*, are examined; and divers of them Censured.

And lastly, A new Way of Amputation, and a speedier convenient Method of curing Stumps, than that commonly practised, is with divers other useful matters recommended to the Military Chirurgeon,

IN TWO LETTERS:

The one to his most Honoured, *James Pearse Esq;* Chirurgeon to His Royal Highness the Duke of York, and Chirurgeon General to His Majesty's Navy Royal.

The other, to Mr. *Thomas Hobbs*, Chirurgeon in London.

By JAMES TONGE.

L O N D O N,

Printed for *J. Martyn*, Printer to the Royal Society, at the Bell in St. Paul's Church-yard. 1679.

by four or five strong stitches and, having so done, clap a dossil of lint into the inferior part, that one passage may be open for any blood or matter that may lodge between. Then lay on a common Defensative Ext. Bole, Sang. Dracon, Mastich, Terre Sigil. cum alb. ovorum & Aceto and thereto gird it close with your cross bandage and other Compresses after the usual manner."

The surgical world must have been ripe for a change from the circular amputation. The flap operation soon came into use in England in spite of the obscure way in which it had been published. A similar operation was practised by Verduyn at Geneva in 1696, by Sabourin at Geneva, and by Garengnot in Paris, each of whom claimed priority.

Some facts about James Yonge are still available, for he was a remarkable person. Born in 1646, he was the son of John Yonge, a surgeon, and was educated at the Plymouth Grammar School until, in 1656, he was apprenticed to Silvester Richmond of Liverpool, who was surgeon to the King's ship, the *Constant Warwick*. He was appointed surgeon's mate to the *Montagu*, a third-rate of 52 guns (formerly the *Lime*), which was built at Portsmouth in 1654 by Mr. Tippetts. Samuel Pepys says of it on May 2, 1661, "We and our wives all to see the 'Montagu' which is a fine ship." It took part in the bombardment of Algiers in 1662 and was afterwards paid off. Yonge then acted as assistant to an apothecary at Wapping for four months, and subsequently helped his father in practice at Plymouth. In February, 1663, he went to Newfoundland in the *Reformation*, and visited the west coast of Africa in 1664. During a second voyage in 1665 his ship was captured by the Dutch, and he was kept prisoner at Amsterdam until September, 1666. He then returned to Plymouth, where he again practised, but in September, 1668, he revisited Newfoundland, returned to England, and settled at Plymouth in 1670. Here he was appointed Surgeon to the Naval Hospital at a salary of five shillings a day, and in 1674 he was Deputy Surgeon General of the Navy. He was Mayor of Plymouth in 1694, and in 1702 he came to London and presented himself for examination at the Royal College of Physicians, although he had long practised on the licence of the Bishop of Exeter. Of this examination he left an interesting account. It was conducted in Lincoln's Inn Fields at the house of the President, Sir Thomas Millington, and was an ordeal of no perfunctory character. "At four o'clock it began and they held me to it till half past five. When they had done they desired me to withdraw and in 4 minutes called me in and told me they were satisfied with my abilities." When the result had been made known to him, "We sat two hours drinking good ale and claret and talking sometime of news and sometime of art." He corresponded with Robert Hooke, the great experimental philosopher who was then Secretary of the Royal Society. Hooke published two of his letters in the *Lectiones Cutlerianæ*; the first describing the fatal symptoms caused by a bullet swallowed into the lungs is dated 1676; the second on May 5th, 1678, contains his own observations and opinions concerning natural fountains and springs. Both are included in *Early Science in Oxford*, vol. viii, published by Mr. R. T. Gunther in 1931. He was elected a Fellow of the Royal Society on Nov. 3, 1702, and in 1707 he embalmed the body of Sir Cloudisley Shovell, the Admiral who was washed up half-drowned on the beach of Porthellick Cove after his ship, the *Association*, had been wrecked on

the Bishop Rock between Land's End and the Scilly Islands. The Admiral was found by a woman who allowed him to die for the sake of a valuable emerald ring which he was wearing. Yonge died on July 25, 1721, and was buried in St. Andrew's Church, Plymouth.

The flap operation thus introduced soon underwent many modifications. The single flap was replaced by double flaps, and the method, at first restricted to the leg, was extended to the other limbs. Liston and Fergusson were its great advocates in England, Liston being so whole-heartedly devoted to it that in his *Elements of Surgery* he entirely omits mention of the circular amputation, though he describes it somewhat grudgingly in his *Practical Surgery*. Speed was of the utmost importance in amputating before the use of anæsthetics, and in the hands of master surgeons like Liston and Fergusson the flap operation was a little quicker than the circular amputation, though in the naval and military services, especially when the operation was required after gunshot wounds and the shattering accidents which were not uncommon on board ship, the circular method remained in favour.

(To be continued)

## STRANGULATED HERNIA: A REVIEW OF 1487 CASES.

BY CLAUDE FRANKAU,

SURGEON, ST. GEORGE'S HOSPITAL, LONDON.

THE case records upon which the subject-matter of this paper is based were obtained from a large number of surgical centres for the purpose of a collective investigation instigated by the Association of Surgeons of Great Britain and Ireland.

In the case of inguinal and femoral hernia the criterion of strangulation has been the presence of some or all of the symptoms of absolute constipation, pain, and vomiting, and—*more important*—evidence at operation of some interference with the blood-supply of the contents of the sac. Cases of simple obstruction in a hernia without strangulation, cases treated by taxis alone, and cases in which the hernia spontaneously reduced under the anæsthetic have been excluded. In considering umbilical hernia the same criterion has been used as far as possible, though some difficulty has been experienced in excluding cases in which obstruction rather than strangulation was present.

## INGUINAL HERNIA.

The total number of cases under review is 654, of which 581 were in males and 73 in females.

**Age Incidence and Mortality.**—*Table I* shows the age incidence and mortality at various ages: an exceptionally large number of cases is recorded among infants; in other respects the incidence conforms with the usually

Table I.—STRANGULATED INGUINAL HERNIA.

## AGE INCIDENCE AND MORTALITY.

Males, 581 (70 died). Females, 73 (13 died).

YEARS	CASES	DEATHS
0-2*	38	2
2-5	7	0
6-10	5	0
11-20	27	1
21-30	66	2
31-40	54	2
41-50	100	7
51-60	124	23
61-70	142	23
71-80	75	17
Over 80†	16	6

\* Youngest, 7 days (died). † Oldest, 90 years (recovered).

accepted figures—the greater number occurring in the periods after 40. The mortality rises steadily with increasing age, and among women (17·8 per cent) is considerably higher than among men (12·0 per cent).

**Special Types of Hernia.**—The types of hernia met with were as follows: oblique 636, direct 18; of these, 31 were recurrent herniæ (oblique 27, direct 4), and of the recurrencees one direct hernia strangulated on its fourth recurrence, having been strangulated twice before. In one case the patient, an infant, had a bilateral

strangulated oblique inguinal hernia, and in one case in addition to the strangulation of an inguinal hernia a similar condition was present in a femoral hernia.

**Duration.**—In 621 cases in which this information was recorded the duration of the hernia before strangulation was as follows: Less than one year, 63 (including 31 infants of less than one year); more than one year, 491; strangulation was the first sign of the presence of a hernia in 67. Primary strangulation, although slightly more common in the third and fourth decades, occurred in patients of all ages except in infants.

The duration of symptoms before operation is shown in Table II, together with the death-rate for the various periods of time. It will be seen that, apart from medical complications and accidents, the mortality in the first twenty-four hours is comparatively low, but that after this period a rapid rise in death-rate obtains. Of the 20 deaths occurring in the first three periods (up to eighteen hours), 12 died from conditions consequent on or aggravated by the operation, and not as an immediate result of the strangulation, the cause of death in these cases being: Pneumonia 4, cardiac lesion 2, uræmia 2, hæmatemesis 1, embolism 1, chloroform 2.

Table II.—STRANGULATED INGUINAL HERNIA.  
DURATION OF SYMPTOMS BEFORE  
OPERATION, WITH DEATHS.

DURATION	CASES	DEATHS
Hours		
0-6	125	4
7-12	149	11
13-18	35	5
19-24	104	5
25-36	19	6
Days		
2	61	16
3	29	8
4	30	12
5	11	4
6	3	0
7	5	1
8	1	0
14	2	1
Duration not stated	80	10
Total ..	654	83

**Symptoms.**—The symptoms noted in detail in 606 case sheets show no remarkable feature: pain, vomiting, and constipation were noted in 424 cases; pain alone or pain and constipation in 168 cases, of which 34 were instances of strangulated omentum only. Persistent vomiting as the sole symptom was noted

in 14 cases, including 3 of strangulated intestine in infants, 3 of strangulated omentum in adults, and 4 of strangulation of large intestine or cæcum.

In 20 cases definite *stercoraceous vomiting* was recorded, and of these no fewer than 15 recovered after operation: this appears to cast some doubt on the statement, so frequently made, that stercoraceous vomiting is always a presage of a fatal issue in cases of intestinal obstruction.

A grave degree of *shock* was noted in 6 cases—all in young men in whom the hernia had come down for the first time and presumably due to the sudden stretching of the sensitive parietal peritoneum at the neck of the sac.

**Site of Strangulation and Contents of Sac.**—The site at various ages is indicated in Table III: the comparative frequency of strangulation at the external ring in infants and the gradual increase of frequency of this site with advancing years is noteworthy. Besides those recorded in the table, in 5 cases strangulation resulted from the presence of a band within the sac.



The contents of the sac are given in *Table IV*, which is self-explanatory.

*Table III.*—STRANGULATED INGUINAL HERNIA.  
SITE OF STRANGULATION IN 497 CASES.

AGE	NECK OF SAC	EXTERNAL RING
0-2	17	17
2-10	3	1
11-20	27	0
21-30	47	6
31-40	25	11
41-50	70	7
51-60	78	15
61-70	74	18
71-80	49	16
Over 80	11	5
Total ..	401	96

One case alone calls for comment, that of a child aged 3 months, where the contents of the sac was strangulated omentum only.

**Unusual Types.** — Unusual types of hernia occurring in the total series of 654 cases were as follows :—

1. *Interstitial hernia*: 6 cases (0·9 per cent). In 3 cases the testis was imperfectly descended and in 2 it was normal. The remaining case occurred in a woman. (The site of strangulation in these cases was in the secondary sac.) All recovered.

2. *Littre's\* hernia*: 4 cases

(0·6 per cent). In one instance the Meckel's diverticulum was resected, in the others it was replaced without treatment. All recovered.

*Table IV.*—STRANGULATED INGUINAL HERNIA. CONTENTS OF SAC.

Small intestine .. ..	433
Small intestine and omentum .. ..	61
Small intestine and cæcum and appendix .. ..	1
Small intestine and appendix .. ..	3
Small intestine and sigmoid .. ..	2
Small intestine and uterus .. ..	1
Small intestine and bladder .. ..	6
Large intestine .. ..	6
Large intestine and omentum .. ..	9
Cæcum and appendix .. ..	17
Appendix .. ..	1
Appendix and omentum .. ..	1
Omentum only .. ..	41
Meckel's diverticulum .. ..	4
Uterus .. ..	1
Appendix epiploica .. ..	1
Sliding hernia of cæcum .. ..	8
Contents of sac not recorded .. ..	58

3. *Richter's hernia*: 15 cases (2·2 per cent). Of these, 6 were treated by simple herniotomy and all recovered: 3 required local repair to the strangulated gut in addition—of these, 2 recovered and 1 died; 1 case was treated by enterostomy and subsequent resection with success. In 2 cases a primary resection was successfully performed. In 3 the abdomen was opened for the treatment of intestinal obstruction of undiagnosed cause, with 2 deaths.

\* This type of hernia was first described by Alexis L. Littre (b. 1658) in a paper entitled "Observation sur une nouvelle Espèce de Hernie" published in 1700 (*Mém. de l'Acad. roy. des Sci.*, Paris, 1700). There is no justification for the addition of an accent on the terminal *c* of this distinguished surgeon's name, although this error has been continued in countless text-books and articles during the last hundred years.

4. *Maydl's hernia*: 4 cases (0·6 per cent), with 3 recoveries. Resection of the re-entering loop was required in one patient, who survived.

5. *Reduction en masse*: 4 cases (0·6 per cent) were reported, with 3 deaths. In all cases a resection had to be performed.

**Operations.**—In *Table V* details are given of the operations performed in 651 out of the 654 cases under review: 3 patients died before any operation could be undertaken.

*Table V.*—STRANGULATED INGUINAL HERNIA. DETAILS OF OPERATIONS PERFORMED.

	CASES	DEATHS
Herniotomy without special operative treatment to bowel	578	46
Herniotomy with precautionary lateral anastomosis ..	2	0
Herniotomy with doubtful gut left unreduced ..	2	1
Primary drainage of gangrenous loop ..	10	7
Local repair of damaged bowel wall ..	15	4
Local repair of damaged bowel wall with lateral anastomosis ..	1	0
Primary resection of gangrenous bowel ..	30	14
Primary resection of gangrenous bowel with temporary enterostomy ..	3	2
Laparotomy for reduction <i>en masse</i> ..	4	3
Laparotomy for undiagnosed Richter's hernia ..	3	2
Laparotomy for double hernia, the side strangulated being in doubt ..	2	1
Local drainage of abscess in sac ..	1	0
Died before operation could be undertaken ..	3	3
Total ..	654	83

Secondary enterostomy was also performed subsequent to the initial operation in 4 cases, with 3 deaths.

Among the 578 cases of *simple herniotomy* where no special treatment of the affected bowel was necessary the following points are of interest:—

1. *The Omentum.*—Congested, strangulated, or adherent omentum was removed in 69 cases with no ill effect or subsequent complication. The risks of spreading thrombosis, hæmorrhage, or necrosis after this proceeding appear to be minimal.

2. *The Testicle.*—In 24 cases a testicle was removed—in 15 instances because it was imperfectly descended, and in 9 owing to damage to the blood-supply. One imperfectly descended testicle was grafted into the patient after removal.

3. *The Bladder.*—Accidental injury to the bladder occurred on 3 occasions: in each the injury was detected at the time and suitably treated with success. No case of undiagnosed injury to the bladder is recorded.

4. *Operation on the Wrong Side.*—In one case, of a man with a large tense hydrocele filled with altered blood on one side and a small strangulated hernia on the other, the hydrocele was explored under the impression that it was a strangulated hernia and was the cause of the symptoms. The patient's condition did not allow of any further operation and he died.

5. *Reduction en Masse at the Time of Operation.*—This accident occurred in one case—it was not detected—with a fatal result.

6. *Fæcal Fistula.*—In one case a fæcal fistula developed at the site of the herniotomy wound; this closed spontaneously.

7. *Internal Strangulation*.—In 2 cases internal strangulation by a band occurred on the sixth and tenth days respectively after the original operation. This condition was successfully relieved by laparotomy in one case.

In considering the 73 cases in which *something more than a simple herniotomy* was required it will be seen that enterostomy, whether primary, secondary, or precautionary (as after excision), is associated with a very high mortality, as must be the case in dealing with desperate conditions. It would, however, appear possible that more cases could be saved if a temporary enterostomy, a foot or more above the site of strangulation, were performed more frequently as a primary measure in cases showing considerable distension of the intestine, and also if a similar method were adopted as an additional measure in cases unfit for resection and treated hitherto by simple drainage of the gangrenous loop.

Of the *enterostomy operations*, one, a secondary enterostomy, is of particular interest. The patient, an infant of 2 months, came under treatment for bilateral strangulated hernia; a double herniotomy was performed with success, but unfortunately on the fourth day the hernia recurred on the right side and again strangulated. The child's condition being desperate, an enterostomy was performed by means of cautery puncture of the serotal contents, the manœuvre being based on the practice in vogue among certain native tribes. The child recovered.

Little comment is required concerning the other operations performed: the cases of resection will be considered later in the paper together with those necessitated in the treatment of other forms of hernia.

It is of interest to note that where a simple herniotomy alone was necessary the mortality was 7·9 per cent, whereas as soon as any further method of treatment was required owing to injury to the bowel wall, etc., the mortality rose to slightly over 50 per cent.

**The Cause of Death.**—The cause of death in the 83 fatal cases can be fairly divided into two groups: (1) Causes directly consequent on the condition for which the operation was performed; (2) Causes due to complications which may follow any operation or due to intercurrent maladies.

In Group 1 the following causes of death were noted:—

Ileus	..	..	..	6
Toxæmia	..	..	..	17
Peritonitis	..	..	..	13
Shock	..	..	..	18
Cause not definitely stated	..	..	..	5
				59

In Group 2 deaths were assigned as follows:—

Pneumonia	..	..	..	12
Bronchitis	..	..	..	3
Cardiac lesions	..	..	..	1
Uræmia	..	..	..	2
Hæmatemesis	..	..	..	1
Pulmonary embolism	..	..	..	3
Anæsthetic	..	..	..	2
				24

The gross mortality for the 654 cases under review was 12·6 per cent.

## FEMORAL HERNIA.

Case records were received of 680 patients, of whom 144 were males and 536 females.

## Age Incidence and Mortality.

—Table VI shows the age incidence and mortality at various ages: the rise in mortality with age is practically the same as in inguinal hernia, but the mortality among men is in the case of femoral hernia far higher than in women (males 22.9 per cent, females 10.2 per cent), whereas the reverse applied in inguinal hernia.

**Duration.**—In 18 cases the hernia when strangulated had recurred after a former operation, and in 2 instances an operation for strangulation had been performed previously. *The duration of the hernia prior to strangulation*

Table VII.—STRANGULATED FEMORAL HERNIA.  
DURATION OF SYMPTOMS, WITH DEATHS.

DURATION	CASES	DEATHS
Hours		
0-6	58	3
7-12	72	3
13-18	25	3
19-24	114	1
25-36	24	12
Days		
2	92	9
3	79	18
4	49	8
5	30	8
6	10	2
7	24	5
9	3	2
10	1	1
14	4	1
21	2	0
30	1	0
42	1	0
Duration not stated	91	12
Total ..	680	88

Table VI.—STRANGULATED FEMORAL HERNIA.

AGE INCIDENCE AND MORTALITY BY AGE.  
Males, 144 (33 died). Females, 536 (55 died).

YEARS	CASES	DEATHS
11-20*	6	0
21-30	29	2
31-40	77	3
41-50	126	9
51-60	155	15
61-70	194	37
71-80	77	16
Over 80†	16	6
Total ..	680	88

\* Youngest, 12 (recovered). † Oldest, 86 (recovered).

was recorded in 550 cases; in 84 patients it had been present for less than one year: in 358 for more than one year, and in 108 strangulation was the first sign of the presence of a hernia.

In Table VII is shown the *duration of symptoms* before operation and the death-rate for the various periods of time. Here also, as in inguinal hernia, the mortality is low for the first twenty-four hours and then rises rapidly. It will be observed that in a very much larger number of cases than in inguinal hernia the symptoms had been present for forty-eight hours or more; this is difficult to explain, as it would be expected that the rigid femoral canal would produce symptoms of strangulation of sufficient urgency to call for earlier intervention. In a certain

proportion (30 per cent) of these cases of long-standing strangulation the contents of the sac was omentum or gut and omentum, in which latter case

the omentum serves to some extent to protect the gut from injury. Similarly the hernia was of the Richter type in 44 cases in which the damage was localized to a small area of the bowel wall, and the symptoms were consequently less severe at first. A large number of cases, however, still remain in which it is difficult to explain the delay in the patient's seeking surgical aid unless the tightness of the strangulation is actually less acute than appears to be the case from an anatomical point of view. An argument against the latter suggestion is the fact that in femoral hernia a considerably higher percentage of cases required special operative treatment for damage to the bowel wall than in inguinal hernia and the resection-rate was considerably higher.

**Symptoms.**—The symptoms recorded in 615 cases are almost exactly comparable with those occurring in strangulated inguinal hernia. *Pain, vomiting, and constipation* was noted in 493 cases; *pain and constipation* in 117 cases, of which 48 were cases of strangulated omentum. *Persistent vomiting* without other marked symptoms was the feature in 3 cases, and in one instance the vomiting persisted for six weeks before operation for a strangulated hernia of the sigmoid and small intestine, which were both described as being 'extremely congested'.

*Visible peristalsis* was noted in one case of strangulation of small intestine; the gut at operation was found to be gangrenous. In one case the most pronounced symptom was the presence of an *abscess* in the sac, which after incision developed into a faecal fistula; in one case the patient came under observation with a *faecal fistula* at the site of the hernia.

*Stercoraceous vomiting* was noted in 26 cases, with 9 deaths and 17 recoveries: the high rate of recovery, as in the inguinal cases, is remarkable.

**Site of Strangulation and Contents of Sac.**—A note was made of the site of strangulation in 525 case sheets, namely:—

Neck of sac and structures surrounding it	..	320
Neck of sac and Gimbernat's ligament	..	29
Gimbernat's ligament	.. ..	174
Saphenous opening	.. ..	1
Adhesions in sac	.. ..	1

It will be seen from these figures that in the larger number of cases strangulation is produced by the neck of the sac and the structures in its immediate neighbourhood, of which the 'deep crural arch' is probably the most important. In old-standing cases, where the neck of the sac has been stretched to some extent by the long-continued presence of a hernia, two other anatomical structures become of importance, i.e., Hey's ligament or the upper edge of the saphenous opening, over which the hernia turns as it passes upwards and outwards after traversing the crural canal, and Gimbernat's ligament, the sharp edge of which acts almost as a knife-edge in its pressure against the protruded gut. These two ligaments act together, the hernia being first fixed by Hey's ligament, and then as it increases in size being pressed against the edge of Gimbernat's ligament. It is open to question whether Gimbernat's ligament is ever the sole cause of strangulation, or whether it is necessary to divide this ligament in any case in which the inguinal method of approach is used: free division of the neck of the sac

from above and below and of Hey's ligament should be sufficient to allow of reduction. Division of Gimbernat's ligament adds greatly to the difficulty of satisfactory repair of the canal.

The contents of the sac are given in *Table VIII*. The only point requiring comment is the large proportion of cases in which omentum was present either with or without intestine as compared with inguinal hernia—namely, 273 cases out of 639 in the case of femoral and 112 cases out of 616 in the case of inguinal hernia. Where omentum and intestine were both contained in the sac it is remarkable that in a considerable proportion of the cases the omentum was more severely affected than the gut, which had presumably been protected by the presence of the omentum.

*Table VIII*.—STRANGULATED FEMORAL HERNIA. CONTENTS OF SAC.

Small intestine .. ..	339
Small intestine and omentum ..	164
Small intestine and appendix ..	4
Small intestine and sigmoid ..	2
Small intestine and bladder ..	3
Large intestine .. ..	4
Large intestine and omentum ..	1
Cæcum .. ..	2
Cæcum and appendix .. ..	4
Appendix alone .. ..	4
Omentum alone .. ..	106
Omentum and bladder .. ..	2
Meckel's diverticulum .. ..	1
Appendix epiploica .. ..	3
Contents not stated .. ..	41
Total .. ..	680

#### Special Types of Hernia.—

*Little's hernia*: In the one case in which a Meckel's diverticulum was strangulated a successful result followed excision of the diverticulum.

*Richter's hernia*: 77 cases of this type of hernia were noted, forming an unexpectedly high percentage (11.3 per cent) of the total number. Of these, 62 recovered and 15 died. In 75 cases the small intestine was involved, in 1 the caput cæci, and in 1 the sigmoid. Thirty-nine cases were treated by simple herniotomy, with 5 deaths: in 19 cases, in addition to the herniotomy, local repair was required to the wall of the intestine; of these, 18 recovered and 1 died. Resection of the damaged intestine was necessary in 11 cases, with 4 deaths. Six cases were treated by exploratory laparotomy for an undiagnosed acute intestinal obstruction: of these, 4 died. One case was operated on under the impression that the condition was an acute abscess, perforation having occurred into the sac; this patient eventually recovered. One case was moribund on coming under observation and died without operation. It is noteworthy that approximately 60 per cent of the cases of Richter's hernia did not come under treatment until the condition had been present for forty-eight hours or more.

**Operations.**—The various operations performed in 679 cases are shown in *Table IX*. One patient was moribund on admission to hospital and no operation could be undertaken. The records do not always indicate clearly the type of operation that was performed or the mode of access, but in

616 cases in which a definite statement is made the following methods were employed :—

Femoral route (Lockwood's operation, etc.)	..	285
Inguinal route (Lotheisen type of operation)	..	319
Hey Groves's operation .. .. .	..	12

As is shown by the figures, the operation by the femoral route still has its advocates who claim that for a life-saving operation this method is the better as it is simple and quick. The comparative simplicity and rapidity of the operation are, however, outweighed by the greater certainty of cure given by the inguinal operation, and more especially by the fact that with this latter operation the strangulated intestine can be examined more adequately and a resection can be performed without making any further incision.

It may be noted that on 24 occasions, in addition to an operation by the femoral route, a laparotomy had also to be performed in order to inspect doubtful gut which had slipped back during the operation, or to remove an inflamed appendix or to carry out a resection: these additional operations would not have been necessary if the inguinal route had been utilized.

*Table IX.*—STRANGULATED FEMORAL HERNIA. DETAILS OF OPERATIONS PERFORMED.

	CASES	DEATHS
Herniotomy without special operative treatment to bowel	551	42
Herniotomy with precautionary lateral anastomosis ..	3	1
Herniotomy with doubtful gut left unreduced ..	2	2
Herniotomy with temporary enterostomy ..	3	1
Primary drainage of gangrenous loop ..	11	10
Local repair of damaged bowel wall ..	35	4
Local repair of damaged bowel wall with lateral anastomosis ..	4	1
Local repair of damaged bowel wall with temporary enterostomy ..	1	0
Primary resection of gangrenous bowel ..	57	21
Primary resection of gangrenous bowel with temporary enterostomy ..	2	1
Secondary resection after primary lateral anastomosis, gangrenous bowel being left outside abdomen ..	1	0
Laparotomy for undiagnosed acute obstruction ..	7	4
Drainage of abscess in sac with subsequent fecal fistula ..	2	0
No operation: patient moribund ..	1	1
Total .. .. .	680	88

Laparotomy was also performed in 4 cases to inspect intestine which had slipped back during herniotomy, for the removal of an inflamed appendix, etc.

1. *The Omentum.*—Affected omentum was removed at operation on 120 occasions with no apparent ill effect or complication afterwards: as a similar satisfactory result was obtained in the inguinal cases it would appear that the reputed dangers of hæmorrhage, spreading thrombosis, or interference with the transverse colon are negligible.

2. *Empty Sac.*—In 3 cases presenting all the typical symptoms and signs of strangulation, at operation the sac was found to contain nothing further than fluid under tension. This condition, which is not very uncommon, is difficult to explain, but it seems possible that temporary blockage of the

uppermost part of the neck of the sac may have resulted from the apposition to it of a tag of omentum or of a portion of bowel—in the latter case the condition would really be the early stage of a Richter's hernia. On opening the sac from below it is obvious that no contents other than fluid would be found and it would be difficult to detect the original cause of the trouble.\*

3. *The Bladder*.—An injury to the bladder necessitating a local repair was noted in 4 cases, all of which recovered.

4. *Abnormal Obturator Artery*.—The abnormality of an obturator artery running along the free edge of Gimbernat's ligament was noted in 3 cases and dealt with by ligature at the time of operation.

5. *Acute Intestinal Obstruction*.—In one case of simple herniotomy acute intestinal obstruction from an omental adhesion to the site of strangulation on the intestinal wall occurred on the fourteenth day. This was treated by laparotomy with success: a further obstruction occurred on the twentieth day from the same cause, and was again successfully treated by laparotomy with recovery of the patient. In this case at the original operation the sac contained gut only.

The number of cases requiring something *more than a simple herniotomy* was considerably larger in femoral hernia as compared with inguinal (76 in inguinal, 128 in femoral). This difference was almost entirely due to the increased number of cases requiring local repair to damaged bowel wall and to the greater number of resections, and this increase can be attributed to the large numbers of the Richter type of hernia in which severe damage may occur to a portion of the bowel and also to the serious injury produced by the pressure of the sharp edge of Gimbernat's ligament in the ordinary type of hernia. The actual technique of the repair of damage to the intestinal wall varied with the individual case, but mention may be made of the danger of invaginating a constriction ring involving the whole circumference of the bowel. In 5 cases in which this method was adopted intestinal obstruction occurred within a few days, necessitating a further operation in each case and with a fatal result in 2 cases out of 5. It would appear to be a safer proceeding to perform a resection as a primary operation or to exclude the damaged area, after such a repair, by a lateral anastomosis.

The very high mortality obtaining in cases treated by simple drainage of the gangrenous loop suggests that in this condition local drainage is not sufficient, and that some diminution in the mortality might occur if the bowel were drained above the site of the obstruction as well as locally.

The two cases of abscess in the sac are of interest. In one, what appeared to be an acute abscess in the groin was opened, pus was evacuated, and a faecal fistula developed shortly afterwards: this fistula closed without further treatment. In the second case the abscess burst spontaneously before the patient came under observation, and a faecal fistula developed: this condition was treated by laparotomy and exclusion of the affected portion

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\* In a case of this type recently operated on by the inguinal route, on deliberately opening the peritoneal cavity a loop of intestine with a small circular congested area on its antimesenteric border was noted. This bears out the theory that this condition is an early stage of a Richter's hernia.



of gut (æcæum) by lateral anastomosis followed by a resection of the æcæum and a portion of the ascending colon and ileum at a later date with success.

A consideration of the cases of resection is deferred till later in the paper.

The mortality for the cases of simple herniotomy was 7·6 per cent, whereas in cases requiring more than simple release of the strangulated gut the mortality rose to 35·1 per cent.

**The Cause of Death.**—Using the same classification as in inguinal hernia the causes of death were as follows :—

1. Causes directly consequent on the condition for which the operation was performed :—

Ileus	..	..	..	10
Toxæmia	..	..	..	30
Peritonitis	..	..	..	15
Shock	..	..	..	8
Causes not definitely stated	..	..	..	6
Total ..				69

2. Causes due to complications which may follow any operation or due to intercurrent maladies :—

Pneumonia	..	..	..	13
Uræmia	..	..	..	1
Pulmonary embolism	..	..	..	4
Anæsthetic	..	..	..	1
				19

The gross mortality for the 680 cases under review was 12·9 per cent.

UMBILICAL HERNIA.

Case sheets were received dealing with 153 patients, of whom 23 were males and 130 females.

**Age Incidence and Mortality.**—*Table X* gives the age incidence and mortality for various ages: the

*Table X.*—STRANGULATED UMBILICAL HERNIA.

AGE INCIDENCE AND MORTALITY BY AGE.

Males, 23 (12 died). Females, 130 (50 died).

DURATION	CASES	DEATHS
1 day	1	0
31-40 years	5	2
41-50 "	19	1
51-60 "	58	23
61-70 "	50	28
71-80 "	18	7
Over 80 "	2	1
Total ..	153	62

single instance of strangulation of an umbilical hernia in an infant is included, as in spite of the extreme youth of this patient the case appears to have been one of a true strangulated hernia. The maximum rate of incidence is between 50 and 70 years.

Strangulation occurred in *recurrences* after a previous operation in 21 cases, and in 2 instances the hernia had already been operated upon for strangulation.

In only 14 cases had the hernia been present for less than one year; and in 6 of these patients the hernia strangulated at its first appearance.

**Duration.**—The duration of symptoms before operation is given in *Table XI*, together with the deaths for each period. Of the cases with long-standing symptoms in which recovery took place, the contents of the sac consisted of omentum only or of large gut and omentum: no case of strangulation of small intestine of a duration exceeding 2 days recovered.

**Symptoms.**—The symptoms recorded presented no noteworthy feature except that of the 14 patients noted as having *stercoraceous vomiting* before operation no fewer than 8 recovered. This recovery-rate conforms with that found in strangulated inguinal and femoral hernia and helps still further to refute the usually accepted hopeless prognosis of such a condition.

*Table XI.*—STRANGULATED UMBILICAL HERNIA.  
DURATION OF SYMPTOMS.

DURATION	CASES	DEATHS
Hours		
0-6	7	3
7-12	16	1
13-18	8	4
19-24	28	6
25-36	8	3
Days		
2	16	12
3	17	3
4	12	8
5	6	6
6	2	2
7	4	2
14	1	0
Duration not stated	28	12
Total ..	153	62

**Cause of Strangulation and Contents of Sac.**—The cause of strangulation in 136 cases in which this fact was recorded was as follows:—

Neck of sac ..	109
Band within sac ..	6
Saccule within sac ..	18
Implanted filigree ..	3

Strangulation in a saccule within the sac is of importance as the clinical signs of strangulation in the hernia itself may be absent or negligible, especially if the hernia is large and the saccule is small and deeply placed. The existence of this condition, which is not uncommon in umbilical hernia, should therefore always be borne in mind.

The contents of the sac are given in *Table XII*. The most remarkable feature presented in this is the fact that small intestine or small intestine

*Table XII.*—STRANGULATED UMBILICAL HERNIA. CONTENTS OF SAC.

Small intestine ..	48
Small intestine and omentum ..	43
Large intestine and omentum ..	16
Large and small intestine and omentum ..	27*
Omentum ..	12
Contents not stated ..	7

\* In 24 cases the small intestine alone was strangulated.

and omentum formed the contents of the sac rather more than six times as often as large intestine and omentum. It would also appear that where small intestine was found in a sac together with large intestine and omentum

the former had clearly been the last structure to enter the sac, the omentum and colon being as a rule fixed by old adhesions. Further, when small intestine and colon were both contained in a sac the former was almost always alone affected by the strangulation: in 3 only out of 27 cases of this type of hernia was the large intestine involved to any extent.

Of the 12 cases in which omentum alone was strangulated, only 1 died as an immediate result of the operation, death resulting from shock.

Several enormous hernias are recorded: in two the contents consisted of several feet of ileum, the cæcum, and the whole of the colon as far as the sigmoid—both died.

**Special Types of Hernia.**—Two cases of *Richter's hernia* of small intestine are recorded: in one the small intestine alone was involved; in the other omentum was also present—this latter patient died.

**Operations.**—Details of 150 operations are given in *Table XIII*: 3 patients were moribund on admission and died before any operation could be attempted. Various methods of repair were used, but that of Mayo was employed in the majority of the cases; a filigree was used in one case which recurred within two years. Omentum was removed in 46 cases, again without ill effect, as was the case also in inguinal and femoral hernia.

Table XIII.—STRANGULATED UMBILICAL HERNIA. OPERATIONS.

			CASES	DEATHS
Simple herniotomy and repair	..	..	114	33
Primary drainage of intestine—small	..	..	9	9
Primary drainage of intestine—large	..	..	5	5
Primary drainage of intestine—large and small	..	..	1	1
Local repair of bowel wall (colon 3, all died)	..	..	6	3
Local repair of bowel wall with anastomosis	..	..	2	2
Primary resection of gangrenous bowel—small	..	..	11	5
Primary resection of gangrenous bowel—large	..	..	1	1
Primary resection of gangrenous bowel—cæcum and ileum	..	..	1	1
No operation	..	..	3	3
Total	..	..	153	63

Of the cases requiring *more than simple herniotomy* it will be observed that small intestine was affected in far greater proportion than large, and that in every case requiring repair, drainage, or excision of large intestine the patient died. It is also noteworthy that every case of primary drainage of gangrenous intestine died.

The cases treated by excision are considered later. The mortality for simple herniotomy was 28.8 per cent, whereas in cases requiring treatment other than simple release of the strangulation the mortality rose to 76.1 per cent.

**The Cause of Death.**—Adopting the same classification as in the other types of hernia, the fatal cases are grouped as follows:—

1. Causes directly consequent on the condition for which the operation was performed.

Shock	..	..	28
Toxæmia	..	..	17
Peritonitis	..	..	4
Cause not stated	..	..	1
			50

2. Causes due to complications following the operation or to intercurrent maladies.

Pneumonia	..	..	5
Bronchitis	..	..	2
Diabetes	..	..	2
Uremia	..	..	1
Pulmonary embolism	..	..	1
Anæsthetic	..	..	2
			13

The gross mortality for all the cases under review was 41.1 per cent.

### RESECTIONS.

In the consideration of resections of gangrenous intestine only those cases that were performed at the initial operation are included: secondary resections after preliminary drainage, of which 7 were performed with one death, present an entirely different problem, as no obstruction was present at the time of operation.

Of the 1480 operations performed for strangulated hernia, primary resection was required in 105 cases (7 per cent): 33 cases in inguinal, 59 in femoral, and 13 in umbilical hernia. The high rate in femoral hernia corresponds with the long duration of symptoms before operation already noted as occurring in this condition. This point is also indicated in *Table XIV*, which gives

*Table XIV.*—RESECTIONS. DURATION OF SYMPTOMS BEFORE OPERATION.

DURATION	INGUINAL			FEMORAL			UMBILICAL		
Hours	Cases	Recovered	Died	Cases	Recovered	Died	Cases	Recovered	Died
0-6	0	0	0	1	1	0	1	0	1
7-12	1	1	0	1	0	1	0	0	0
13-18	3	2	1	3	1	2	0	0	0
19-24	8	6	2	6	5	1	1	0	1
25-36	2	1	1	3	0	3	1	0	1
Days									
2	7	3	4	10	7	3	1	0	1
3	5	0	5	9	6	3	4	4	0
4	4	1	3	7	5	2	1	0	1
5	0	0	0	4	4	0	0	0	0
6	0	0	0	1	1	0	0	0	0
7	1	1	0	0	0	0	0	0	0
Duration not stated	2	2	0	14	7	7	4	2	2
Total ..	33	17	16	59	37	22	13	6	7

the duration of symptoms in each type of hernia before resection was performed. It will be seen in this table that in the majority of cases requiring resection the symptoms had been present for twenty-four hours or more, and that under twenty-four hours this operation was but rarely required.

Apart from 1 case of resection of gangrenous transverse colon and 2 cases of resection of ileum and cæcum, all of which died, the resections were all of small intestine.

The types of anastomosis used were as follows :—

			Number	Recovered	Died	Mortality
End-to-end	..	..	44	25	19	43.1 per cent
Lateral	..	..	59	35	24	40.6 „ „
End-to-side (ileum to colon)			2	0	2	100 „ „
All types	..	..	105	60	45	42.8 „ „

There seems little difference between the two methods of end-to-end and lateral anastomosis, though the latter shows a higher incidence and a slightly lower mortality. In one case of end-to-end anastomosis mechanical obstruction occurred on the fourth day; this was rectified by a lateral anastomosis.

*Temporary enterostomy* above the anastomosis was performed on 6 occasions, with 2 deaths: it would appear that a further use of this method might diminish the mortality in cases of resection.

The method of access for the resection varied with the type of case: in 30 out of 33 inguinal cases the resection was performed through the herniotomy wound, whereas in the femoral cases in 21 cases out of 59 a separate laparotomy was performed.

## POST-OPERATIVE COMPLICATIONS.

### PULMONARY COMPLICATIONS. ANÆSTHETICS.

Post-operative complications apart from thrombosis and affections of the lungs were few in number and trivial in degree, only two cases of parotitis and two of transient delirium being noted.

*Pulmonary embolism* occurred in 10 cases, with 8 deaths, the earliest case being on the fifth day and the latest on the thirtieth day after operation. No record of the origin of the embolus was given in 8 cases: in 2 the femoral vein was thrombosed—in one case on the same side, and in the other, on the side opposite to that operated on.

Uncomplicated *femoral thrombosis* was noted in 3 cases. Considering the close proximity of the femoral vein to the operation area in both inguinal and femoral hernia, and also that in the majority of cases of strangulation an element of infection is inevitable, the comparatively high rate of incidence of embolism (0.67 per cent) is explicable.

*Post-operative pulmonary complications* of varying degree were noted in 105 cases out of 1480 operations (7 per cent), with 35 deaths—that is, a death-rate of 33.3 per cent for pulmonary complications *per se*, and of 2.3 per cent from pulmonary complications for all cases of strangulated hernia operated on. The exact lesions produced were difficult to determine, but the following list gives them as accurately as possible:—

	CASES	DEATHS
Bronchitis ..	57	6
Bronchopneumonia ..	36	23
Lobar pneumonia ..	10	6
Massive collapse ..	2	0
Total ..	105	35

It was impossible to exclude from the list cases in which bronchitis, etc., were present before the operation, but it can safely be assumed that any antecedent condition was aggravated by the operation.

Five deaths occurred *during the administration of the anæsthetic*: of these 2 can be fairly attributed to the anæsthetic, as death occurred during induction with chloroform; 2 occurred in desperate cases, and can more properly be attributed to the shock of the operation, and in 1 it is recorded that the patient was drowned in his own vomit during the operation.

The various types of anæsthetic employed, with the pulmonary complications occurring in each type, are given in *Table XV*. In this it will be seen that the anæsthetic of choice was ether: under this heading are included all

*Table XV.*—ANÆSTHETICS AND PULMONARY COMPLICATIONS.

ANÆSTHESIA	CASES	PULMONARY COMPLICATIONS	INCIDENCE	DEATHS FROM PULMONARY DISEASE
			Per cent	
General, type not stated	116	7	6.0	0
Ether .. ..	685	52	7.6	20
C.E. mixture .. ..	332	16	4.8	2
Chloroform .. ..	33	5	15.0	4
Gas and oxygen .. ..	68	8	11.0	1
Local .. ..	137	12	8.7	5
Spinal .. ..	108	5	4.5	3
Avertin .. ..	1	0	0.0	0
Total .. ..	1480	105	—	35

cases which were given mainly ether, a case in which C.E. mixture was given for the induction followed by ether being classed as an ether anæsthetic. The incidence of pulmonary complications after ether corresponds almost exactly with the general incidence, and no serious contra-indication to the use of this safe anæsthetic, if inhalation anæsthesia is employed, can be brought forward. The high incidence after chloroform, gas and oxygen, and local anæsthesia can be attributed to the fact that these methods were in all probability used because of a known pulmonary affection in the patient.

It is an accepted fact that pulmonary complications in acute intestinal obstruction are due in the main to the inspiration of highly infected material vomited from the stomach. Some improvement in the very high rate of incidence of and mortality from such complications should therefore be obtainable by careful lavage of the stomach before and after operation, and by the use of local or spinal anæsthesia in preference to any inhalation method.

### SUMMARY.

1. A review of 1487 strangulated inguinal, femoral, and umbilical hernias.
2. The mortality in each group was as follows: Inguinal 12.6 per cent, femoral 12.9 per cent, umbilical 41.1 per cent, and for all cases 15.7 per cent.
3. It was necessary to resect intestine in 105 cases, with 45 deaths.
4. Pulmonary embolism occurred in 10 cases (with 8 deaths) out of a total number of 1480 operations.
5. Pulmonary complications, bronchitis, pneumonia, etc., followed the operation in 105 cases, with 35 deaths.
6. Stercoraceous vomiting was recorded as a symptom in 60 cases, with 40 recoveries and 20 deaths.

## A CASE OF GENERALIZED FIBROCYSTIC DISEASE OF THE BONES.

BY LIEUT.-COLONEL E. W. C. BRADFIELD, I.M.S.,  
PROFESSOR OF SURGERY, MADRAS.

THE patient first came under observation in 1913, when at the age of 6 years he was admitted to the General Hospital, Madras, for a fracture of the right femur. A brief note of his condition was recorded by Mr. Elmslie<sup>1</sup> in 1914.

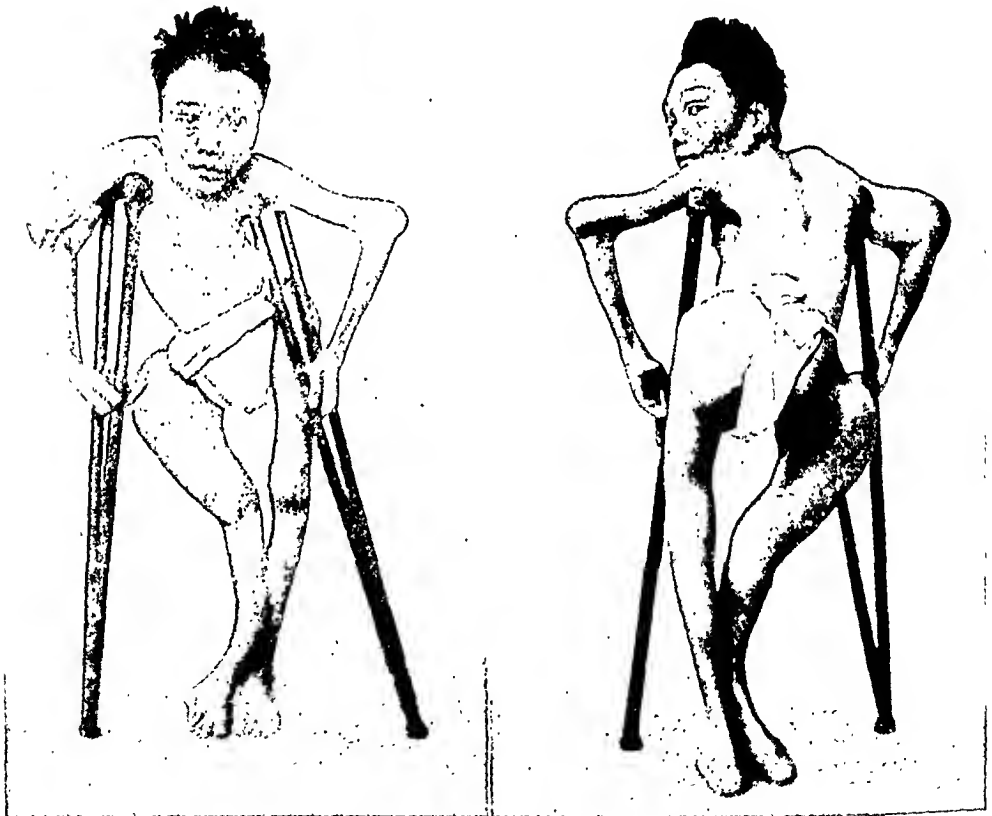


FIG. 159.—Photographs of the patient showing the condition in 1920.

Only a few radiographs were taken at that time, but cysts were found in both humeri, in the shaft of the right femur, which was fractured, the left femur, and left tibia. The skull was asymmetrical but not cystic. The

fractured femur united without incident, as did other spontaneous fractures of the left tibia and left femur during the succeeding eighteen months.

The patient was again seen in 1920 for a deformity resulting from a fracture of the right tibia and for which an osteotomy was performed. The

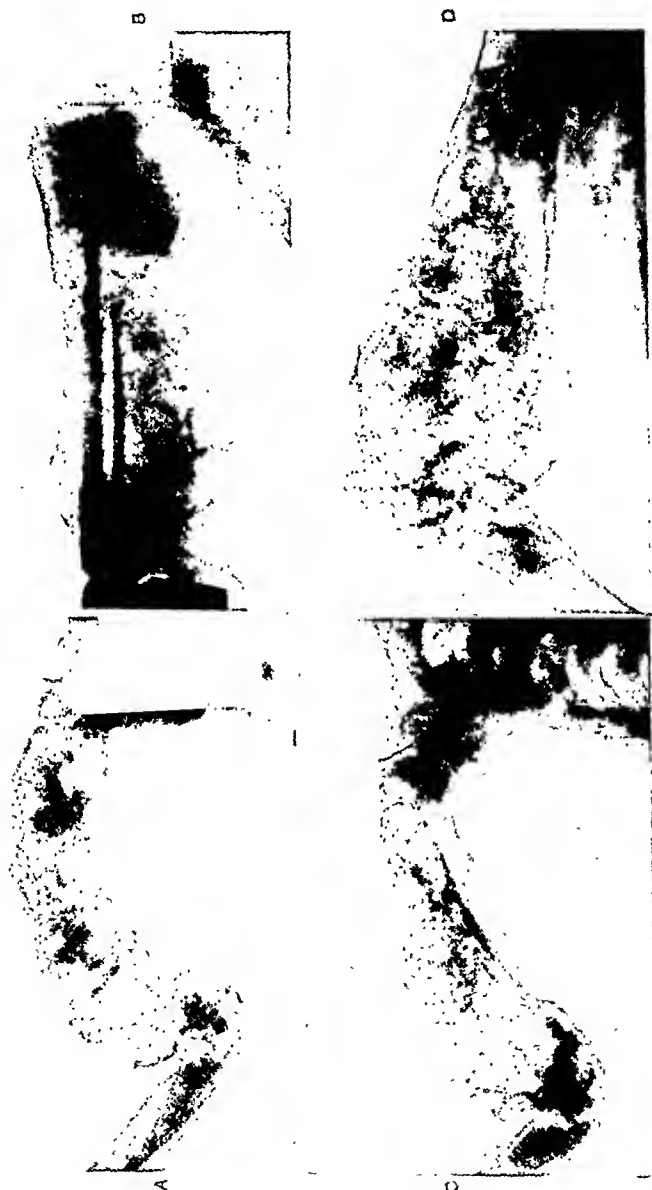


FIG. 160.—X-ray photographs of the right humerus. A, Aug. 4, 1921; B, Aug. 16, 1921, after removal of a portion of the tissue and insertion of a piece of ox-tib; C, June 14, 1922; D, Nov. 26, 1922.

notes of this period record an increased enlargement of the bones, especially of the right and left humeri, which could be dented like a celluloid ball, and were tender on deep pressure. The bones of both lower extremities were harder, did not bend on pressure, and were only slightly tender. No changes were found in the forearms, wrists, hands, feet, or ankles. The condition



of the spine and ribs was not recorded. The operation notes state that the tibia was hollowed out into a number of spaces, crossed by bony trabeculae, in which no definite marrow or medulla could be detected, and that there



FIG. 161.—X-ray photographs of the pelvis. A, Aug. 4, 1921; B, Oct. 30, 1929.

was only a very thin layer of compact tissue. Under the microscope the removed wedge consisted mainly of bony plates enclosing a fibrovascular tissue.

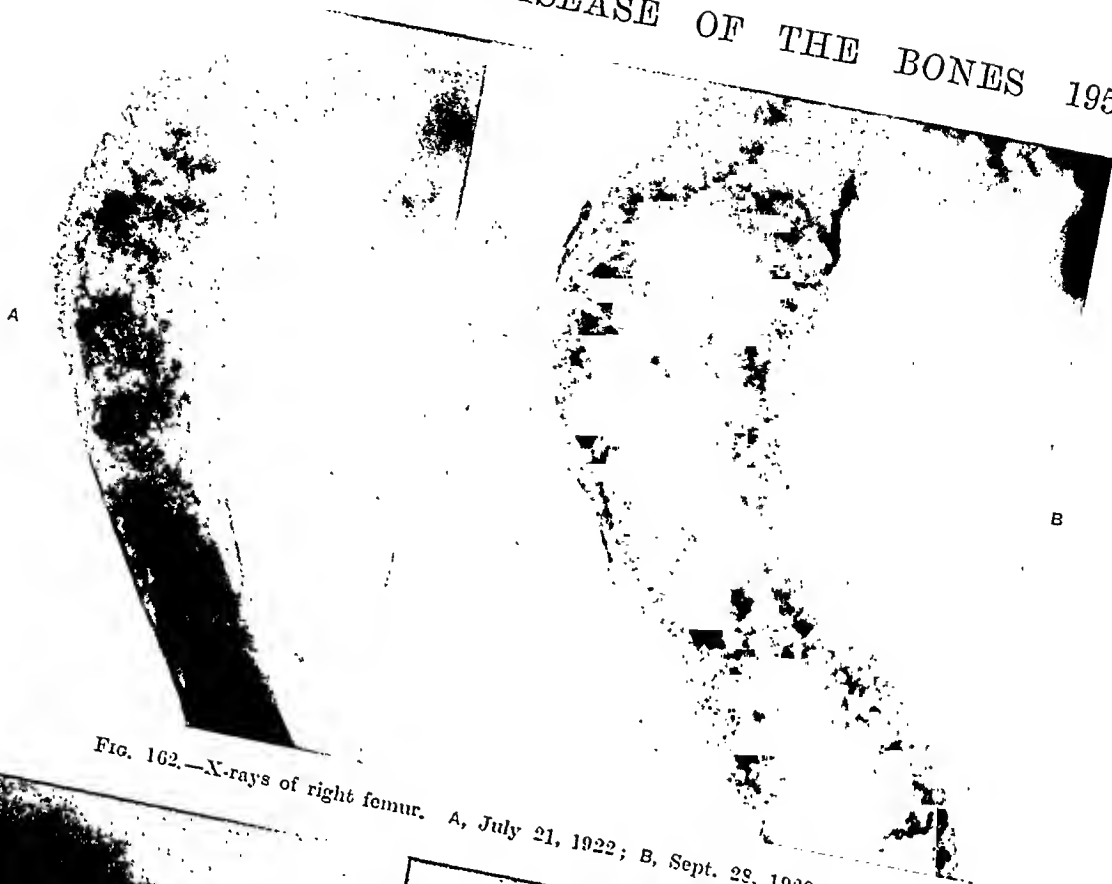


FIG. 162.—X-rays of right femur. A, July 21, 1922; B, Sept. 28, 1929.

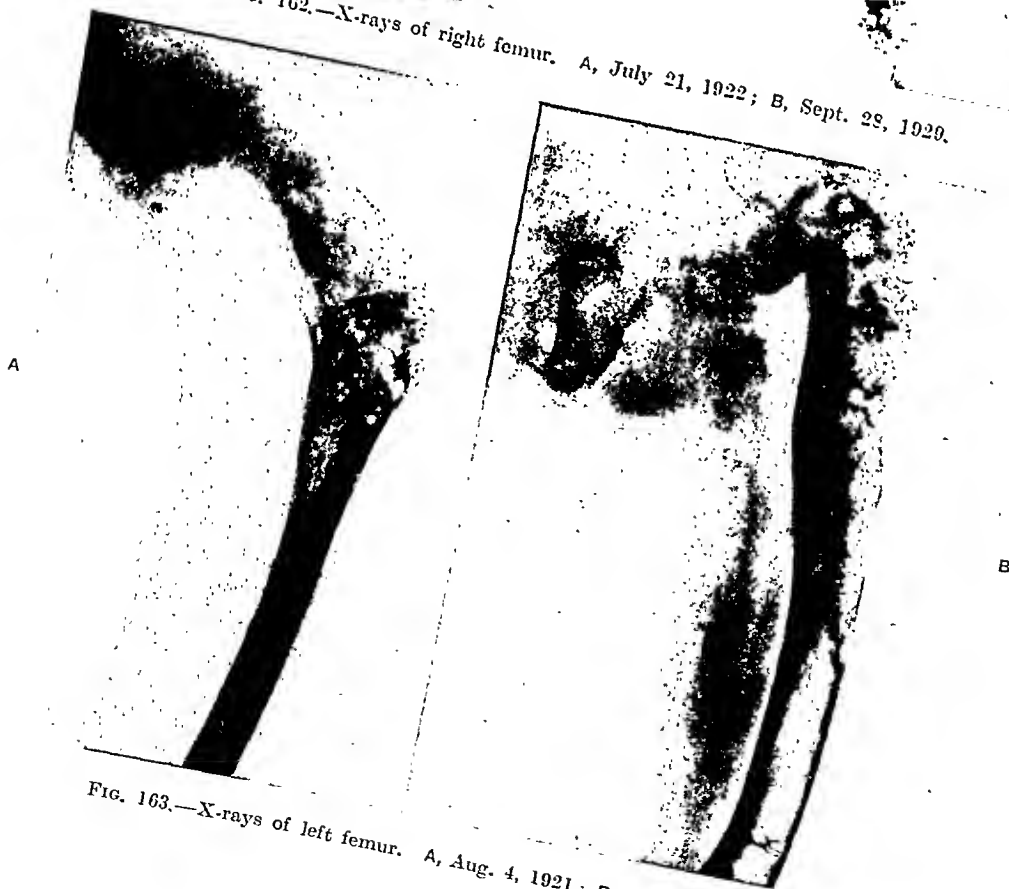


FIG. 163.—X-rays of left femur. A, Aug. 4, 1921; B, Nov. 26, 1929.



FIG. 164.—X-rays of legs. A, Right tibia and fibula, Aug. 4, 1921; B, Right and left tibia and fibula, Sept. 11, 1929.

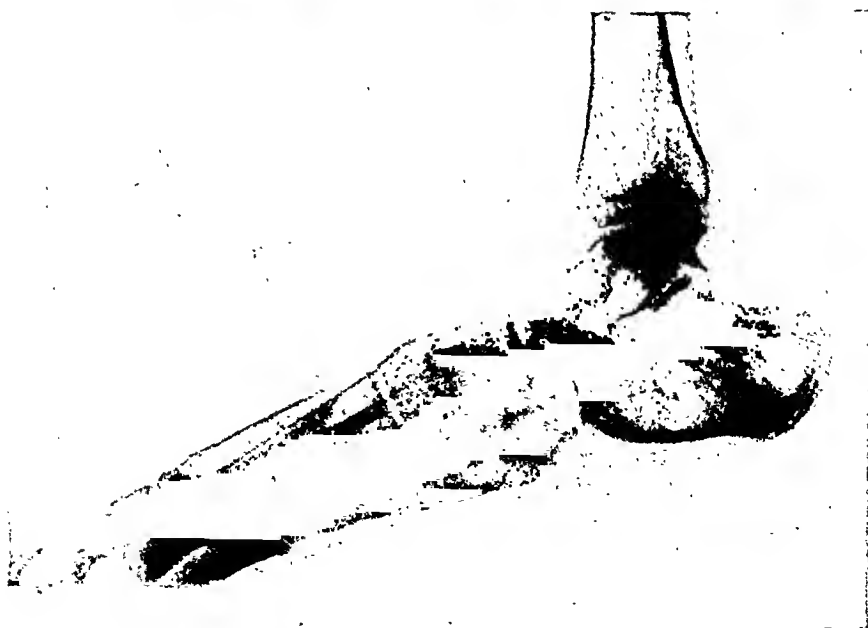


FIG. 165.—X-ray of foot showing a possible cyst in the os calcis.

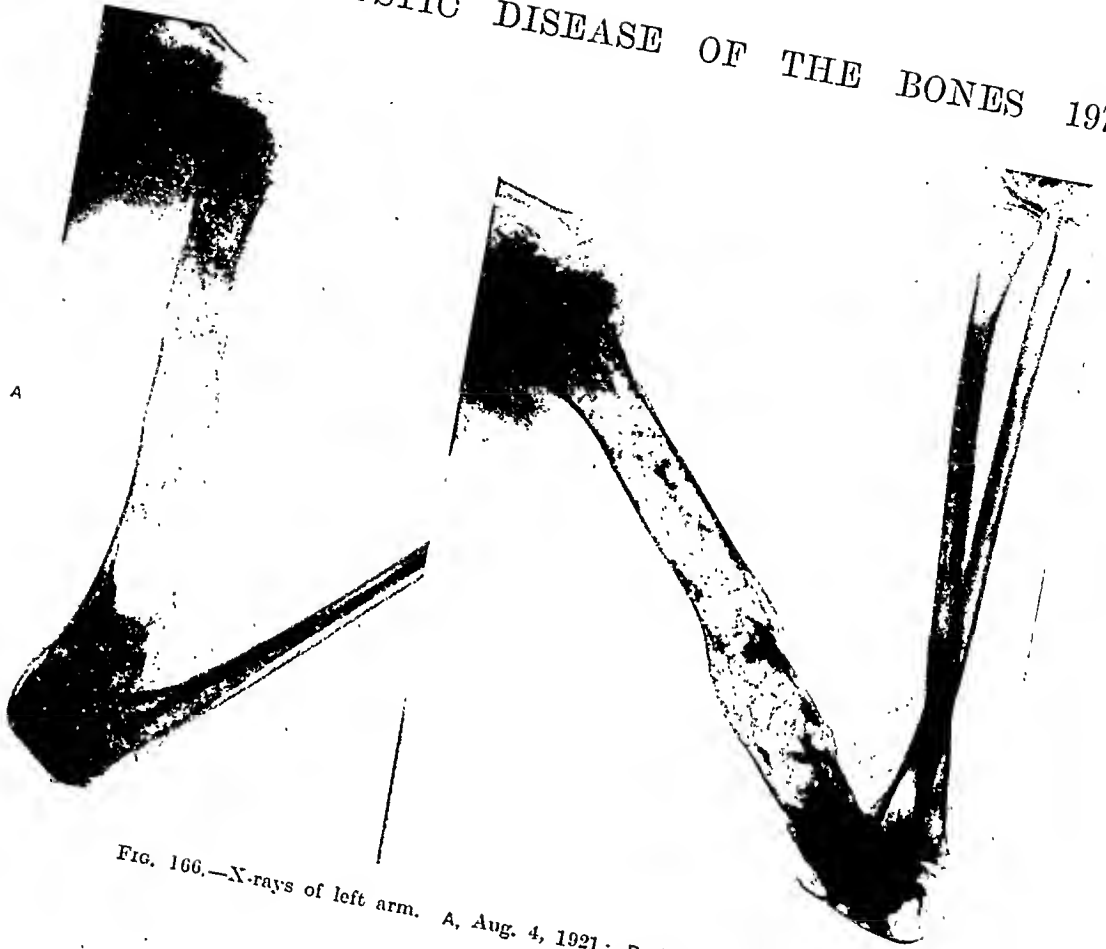


FIG. 166.—X-rays of left arm. A, Aug. 4, 1921; B, Sept. 23, 1929.

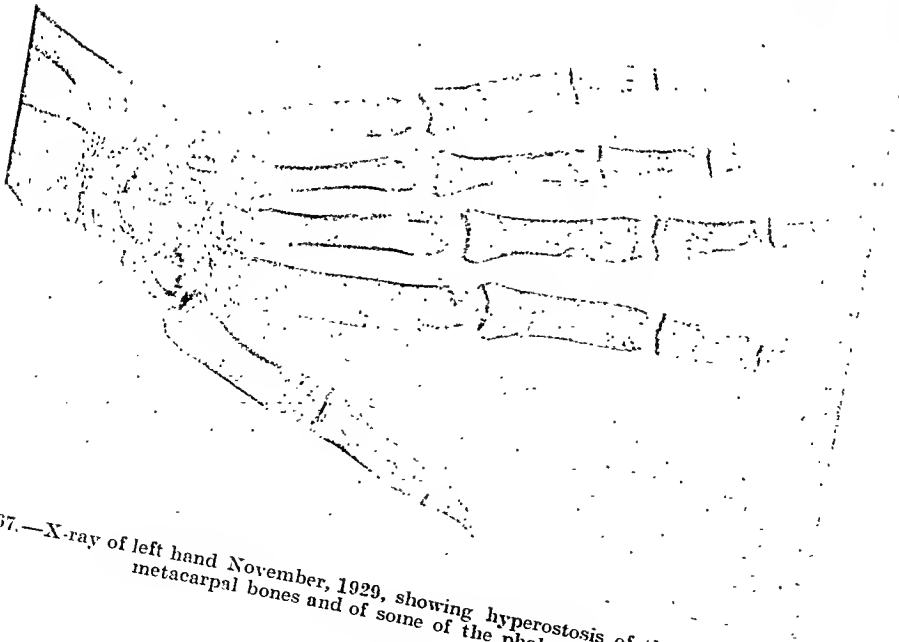


FIG. 167.—X-ray of left hand November, 1929, showing hyperostosis of the 2nd and 5th metacarpal bones and of some of the phalanges.

The patient was again admitted in 1921, and the condition of the bones at that date is shown in the X-rays reproduced (*Figs. 160-166*). The notes record that only the right humerus was tender, painful, and parchment-like. With the idea of stimulating bony growth, a portion of an ox-rib was inserted into the cystic humerus, but although it caused a certain amount of reaction, the graft was gradually absorbed and had no influence on the course of the disease. A portion of cystic bone removed during this operation has been re-examined and is reported on below by the pathologist.



FIG. 168.—X-ray of skull, Nov. 20, 1929.

The patient was again discovered during 1929, and is now a grown, fairly intelligent man, who is considerably handicapped by multiple deformities (*Fig. 159*). The condition of the bones, which are now quite hard, not tender or painful, is seen in the accompanying radiographs (*Figs. 160-169*).

The interest of this case lies in the observations which have been made over a period of seventeen years. The disease appears to have been progressing in the lower extremities between the ages of 6 and 9, when several fractures occurred in the femora and tibiae, and to have still been active in the right



FIG. 169.—X-ray of thorax, Sept. 11, 1929.

humerus up to the age of 18. At the present time it seems reasonable to assume that the disease is no longer active, although no real attempt is being made to replace the cystic tissue by new bone formation. At no time did any pathological investigation throw light on the etiology of the condition. The Wassermann reaction has always been negative; the blood, except for a mild anæmia common in India, has been normal; and no results from careful investigation of the fæces and urine have indicated any relevant metabolic changes. The appearances in the skull remind one very much of those seen in Paget's disease, and the information obtained from the mother that birth was normal suggests that the skull deformity was due to the disease and was probably not recognized in our earlier X-rays.

**PATHOLOGIST'S REPORT (Dr. A. Vasudevan).—**Tissue from A. Boward (Sections 9401, A, B, & C, 98 and 1490). The specimen is a pale bluish cartilaginous mass roughly  $3\frac{1}{2} \times 1\frac{1}{4}$  in., separated into irregular areas by strands of reddish-brown tissue, on which are seen opaque, yellowish, gritty areas of calcified material (*Fig. 170*).

**Microscopical Appearances (Figs. 171–174).—**In the main, the tissue consists of irregularly disposed tracts and islands of osteoid material embedded in a mass of vascular marrow which is in varying stages of fibrosis. The osteoid material is arranged in branching columns or islands, and does not show any Haversian canals, while lamellar arrangement is



FIG. 170.—Portion of bone removed from the right humerus in 1921 (*see text*).



FIG. 171.—Section of tissue from the right humerus. (Low power.) A, Columns of osteoid tissue; B, Cyst containing fibrous tissue.



FIG. 172.—Section of tissue from the right humerus. (Low power.) A, Islands of osteoid tissue; B, Cellular fibrous tissue.

mostly absent or very faint. There are a few lacunæ in which are incarcerated cells. Most of the osteoid masses are lined externally by a definite row of darkly staining cuboidal cells (osteoblasts). In some portions this osteoid tissue is almost absent and its place is taken by large areas of degeneration, some of which show calcification. Between the masses of osteoid material is a vascular fibrous tissue in which are numerous fibroblasts, young blood-vessels,

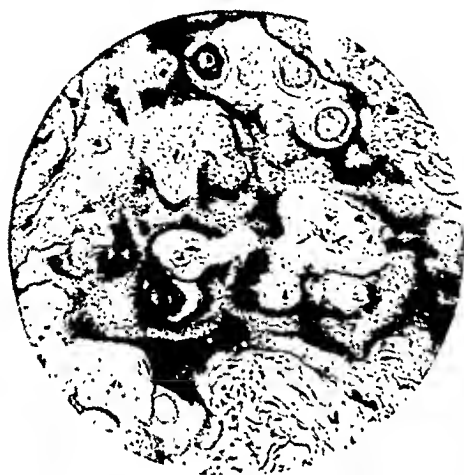


FIG. 173.—Section of tissue from the right humerus. (High power.) Osteoid tissue showing calcification.



FIG. 174.—Section of tissue from the right humerus. (High power.) Cyst in bone, lined with endothelium and containing fibrous tissue.

and a few multinucleated giant cells. This is probably marrow undergoing fibrosis. This marrow tissue is scanty and is well seen in only one of the sections. Numerous cysts of varying size lined with definite flat endothelial cells, some containing blood and others coagulated eosin-staining material, are seen in the fibrous tissue. The cyst formation is not uniform, being marked in some samples of the tissue and almost absent in others.

I am very much indebted to Captain Barnard and the X-ray Institute, Madras, for the pictures which illustrate this case.

### COMMENT.

(R. C. ELMSLIE.)

Further sections of the specimen removed from the humerus submitted to me by Colonel Bradfield showed nothing additional to the tissues described by Dr. Vasudevan except that considerable areas of cartilage were found.

This is a typical case of generalized fibrocystic disease in which the changes consist more in the transformation of bone into fibrous tissue and cartilage than in the formation of cysts. There seem to have been no gross cysts and no evidence of the existence of giant-cell tumours. The extreme



changes in the upper ends of the femora seem to be very characteristic of this clinical type of the disease. In one recent case in which the pathological picture was similar to that described by Colonel Bradfield, investigation showed that the blood calcium and phosphorus were within normal limits, and exploratory operation failed to disclose any parathyroid tumour. Parathyroid tumours have been found in most of the cases in which there is extensive cystic formation. It may be that the fibrosing form is a different disease, or it may be that it represents a late and inactive stage.

Colonel Bradfield's case is of very great interest because it illustrates the progress of the disease over a long period.

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#### REFERENCE.

- <sup>1</sup> ELMSLIE, R. C., *Brit. Jour. Surg.*, 1914, ii, 17.

## **HYPERPARATHYROIDISM: GENERALIZED OSTEITIS FIBROSA.\***

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### **WITH OBSERVATIONS UPON THE BONES, THE PARATHYROID TUMOURS, AND NORMAL PARATHYROID GLANDS.**

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THE purpose of this paper is to set forth the evidence upon which is based the view that generalized osteitis fibrosa (von Recklinghausen) is a disease of endocrine origin. According to this view the changes in the bones are due to hyperparathyroidism, a condition associated with hyperfunction of a parathyroid tumour. Four such cases are described, and in three the effects of removal of a parathyroid tumour are recorded. Four cases of focal osteitis fibrosa are described in order to place on record the absence of any evidence of hyperparathyroidism in this disease. Finally, a case of osteomalacia is recorded. Here the blood chemistry, metabolism, and histology suffice to show that osteomalacia and generalized osteitis fibrosa are unrelated diseases.

### **EFFECTS OF THE PARATHYROID HORMONE.**

The parathyroid glands exert a specific effect upon calcium metabolism. The pioneer work of MacCallum and Vogtlin (1909) demonstrating a reduction of serum calcium in dogs after parathyroidectomy has been abundantly confirmed. It is well established that this condition is relieved by the administration of calcium salts. The idea that it is due to guanidine intoxication remains unsupported by adequate evidence.

Since the isolation by Hanson (1924) and Collip (1925) of the active principle of the parathyroid gland (parathormone) it has been repeatedly demonstrated that the injection of the extract causes an elevation of the serum calcium. Brehme and György (1927) found that parathormone lowers the blood phosphorus and shifts the pH of the blood towards the acid side without affecting the CO<sub>2</sub> combining power of the blood. Reiss (1928) and Aub (1928) found independently that parathormone lowers the level of the blood phosphorus. This appears to be the first action, the elevation of serum calcium following only after a long latent period. Whether the effect on phosphorus is the primary effect of parathormone remains for further work

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*\*Being the basis of an Arris and Gale Lecture delivered before the Royal College of Surgeons, in London, on January 30th, 1931. Received for publication May 12th, 1931.*

to establish. However, the dramatic effect of parathormone is the marked elevation of serum calcium.

The study of the influence of parathormone on the excretion of calcium and phosphorus has demonstrated less striking results than would have been expected from its effect upon the blood. In normal individuals a constant dose of parathormone causes the phosphorus excretion to rise abruptly to a maximum in the first three-day period, while the maximal increase of the calcium excretion follows more gradually. After discontinuing parathormone the phosphorus excretion falls to normal more abruptly than does the calcium (Albright, Bauer, Ropes, and Aub, 1929). This early rapid excretion of phosphorus may account for the primary fall in the plasma phosphorus and is further suggestive evidence of the contention of Greenwald (1924) of the primary influence of phosphorus in regard to tetany.

Parathormone raises the serum calcium in normal persons. There is great individual variation in this response. A dose of 50 units of parathormone daily is usually found to have little influence on the level of the serum calcium, yet it approximately doubles the excretion of calcium and phosphorus. When 100 units a day are given to normal individuals the serum calcium level is raised to an average of 12.5 mgrm. per 100 c.c., and there is an average rise in the urinary calcium excretion to six times the normal level. The source of such large amounts of calcium salts appearing in the urine is of fundamental importance. The evidence available both in animals (Greenwald and Gross, 1925) and in man (Hunter and Aub, 1926) points to the conclusion that *these calcium salts are derived from the bones*.

**Effects of Over-dosage with Parathyroid Extract.**—The work of Collip on dogs (1925) brought to light a new entity—hypercalcæmia. This condition has since been found to occur in man, both spontaneously and as the result of parathyroid over-dosage. In Collip's observations on dogs no ill-effects were noted if the dosage of parathyroid extract was only sufficient to cause a rise in the serum calcium to 14 or 15 mgrm. per 100 c.c. Large doses of the hormone, however, administered five times in the twenty-four hours often caused death within forty-eight hours of the first dose. Though in such cases the serum calcium rose to 20 or 21 mgrm. per 100 c.c., Collip made no claim that the hypercalcæmia was the immediate cause of death. In describing the condition of these animals he states that "the clinical manifestations of hypercalcæmia in dogs appear to be loss of appetite, dullness, drowsiness verging on coma, general atonia, and a failing circulation. The animals are also obviously dehydrated, the blood becomes very concentrated, and it becomes a matter of great difficulty to secure serum from blood samples for analysis even when a high power centrifuge is used. Immediately after death the blood in the heart and great vessels forms a firm clot." Complete investigations of the blood chemistry showed that the inorganic phosphorus rose in the later stages of hypercalcæmia and that there was nitrogen retention. Thus in one dog the blood at death had a phosphorus content of 13 mgrm. per 100 c.c. and a urea content of 111 mgrm.

During our investigations a severe hypercalcæmia was produced in one case by over-dosage with parathormone (Hunter and Aub, 1926). The patient was a man of 60 years suffering from chronic lead poisoning. Parathormone

had been given in increasing doses for seven days, the total amount being 365 units. On the seventh day the serum calcium had risen to 19.8 mgrm. per 100 c.e. It remained at this level for forty-eight hours; then, though all treatment had been discontinued, it fell only very gradually to normal levels during the next three days. The plasma phosphorus dropped to 2.7 mgrm. per 100 c.e., but later it rose to 6 mgrm. per 100 c.e., and this occurred twenty-four hours after the maximum rise in calcium. The non-protein nitrogen showed a rise to a maximum of 95 mgrm. per 100 c.e., this rise occurring two days after the maximum rise in calcium. During the next week it fell gradually to within normal limits. The changes in the physical properties of the blood observed by Collip in dogs suffering from parathyroid over-dosage were absent. Throughout the whole time the patient remained up and about, and, except for slight nausea and loss of appetite, appeared to be normal.

Prolonged over-dosage with parathormone in the dog may give rise to metastatic calcification in the kidneys, lungs, myocardium, and gastrointestinal tract (Hueper, 1927). It is of interest to note that under certain biological conditions hypercalcemia may occur as a normal phenomenon. It has been shown that the serum calcium of certain birds increases very considerably at the period of ovulation. Thus in the female pigeon the rise in the serum calcium begins 123 hours before the formation of the egg-shell. Subsequently, during a period of from three to five days these birds will stand, without trace of adverse effect, a hypercalcemia up to 20 mgrm. per 100 c.e. (Riddle and Reinhart, 1926). Similarly the female cod in the spawning season has a blood calcium varying from 12.7 to 29 mgrm. per 100 c.e., whereas the corresponding figure in the male lies between 9 and 12.5 mgrm. (Hess, Bills, Weinstock, and Rivkin, 1927). This calcium is probably required for the elaboration of the eggs, which are especially rich in that element.

### GENERALIZED OSTEITIS FIBROSA OF VON RECKLINGHAUSEN.

Generalized osteitis fibrosa is a disease which progresses with pain, fractures, and disabling deformities, and is usually fatal. There is widespread pathological resorption affecting all the bones. In addition there are multiple foci of osteitis fibrosa with or without benign giant-celled tumours and cysts. It is a disease entity which I believe to be distinctly different from focal osteitis fibrosa. In the latter there may be one or more focal lesions, the essential point being that outside these lesions all bony tissue is normal.

In a Festschrift written in 1891 in honour of the seventy-first birthday of Virchow, von Recklinghausen gave the first accurate description of the disease entity which now goes by his name. His contribution to the Festschrift is very involved, for he describes more than one bone disease. There can be no doubt, however, that his Cases 5 and 7 constitute the discovery of generalized osteitis fibrosa. It is important for us to notice that he went to the trouble of describing two cases of puerperal osteomalacia (Cases 8 and 10) in order to place on record that "so many facts differentiate Case 7 from true osteomalacia". He describes his Case 7 as "osteitis fibrosa with marked bone softening and multiple osteosarcomata". The patient was

a man of 40, thought to be suffering from osteomalacia. There was a history of eighteen months' bone pain with bowing of the bones and multiple spontaneous fractures. At necropsy the bones were soft and deformed so that with a saw they cut like rotten wood, and the shafts could be cut with a knife using moderate force. All the bones in the skeleton were affected, and seemed histologically to be formed of a tough connective tissue. Brown-red projecting tumours were found in the fibula, iliac crest, and upper and lower jaws.

In our subsequent knowledge of this disease there is ample clinical and pathological evidence to establish the benign nature of these giant-celled tumours. The cells themselves are similar to the osteoclasts normally present in Howship's lacunæ.

Thirteen years after von Recklinghausen first described generalized osteitis fibrosa an important step was made towards solving the problem of its etiology. This was a description by Askanazy in 1904 of a case of generalized osteitis fibrosa associated with a parathyroid tumour. The patient was a woman of 51, complaining of pains in the limbs, and suffering from multiple spontaneous fractures. At necropsy, to the left of the left thyroid lobe there was a tumour which looked almost like a second left thyroid lobe. A parathyroid origin of this tumour was suspected. The femur was fractured in three places, and all the bones of the skeleton cut easily with a knife. Microscopically the process of destruction of old bone was carried out by numerous osteoclasts lying in Howship's lacunæ. Sometimes small bony particles were seen surrounded by osteoclasts, and occasionally these cells alone were seen, singly or in groups, free in connective tissue.

Since its significance was scarcely suspected, the parathyroid tumour mentioned by Askanazy in this case passed unnoticed. However, the description by Erdheim in 1907 of three cases of osteomalacia associated with parathyroid enlargement called forth many other observations of bone disease associated with hyperplasia or tumour formation in the parathyroid glands.

In 1925 Hoffheinz, rejecting all cases where the parathyroid enlargement was indefinite or inadequately described, collected from the literature 45 cases with *measurable* enlargement of one or more parathyroid glands. Of these, 27 were associated with definite bone disease, including 17 of generalized osteitis fibrosa, 8 of osteomalacia, and 2 of rickets. He quoted increases of size up to  $7 \times 2.5 \times 1$  cm. The histological changes were either those of hyperplasia or of adenoma, and it is important to notice that sometimes more than one parathyroid gland was affected.

In cases of generalized osteitis fibrosa renal calculi have commonly been observed, and areas of metastatic calcification have been found in the lungs, stomach, kidneys, and myocardium (Dawson and Struthers, 1923).

**Relation of Parathyroid Tumour to Changes in Bone.**—Amongst pathologists a difference of opinion arose as to whether the parathyroid tumour was primary or secondary in relation to the bone disease. Erdheim himself defended the view that the disease of the skeleton was the primary factor, and that skeletal decalcification created such a demand for calcium that a compensatory hypertrophy of the parathyroid glands occurred. Anatomical evidence against this view was found in the fact that, in generalized osteitis fibrosa, hypertrophy or tumour formation was often confined to one

parathyroid gland, the others remaining apparently normal. On account of Erdheim's investigations the whole question was repeatedly discussed at scientific meetings in Vienna. As early as 1915 Schlagenhauser at such a meeting raised the question whether it was not justifiable to attempt the extirpation of a parathyroid tumour in cases of generalized osteitis fibrosa. Maresch supported this suggestion, but it was not until 1926 that Mandl, of the Vienna school, first performed this operation. In view of the Vienna controversy, it is of great interest that he decided to explore the neck only after treatment by parathyroid transplants and parathyroid tablets had failed to relieve the symptoms. The patient was a man of 38 with a history of pains in the pelvis and lower extremities for five years. Radiograms showed osteitis fibrosa of femora, pelvis, tibia, and ischium. Spontaneous fracture of the femur occurred, and he became unable to stand or sit. Four parathyroid bodies from an accident case were transplanted into the abdominal wall. No improvement occurred. It was therefore decided to explore the neck, though no tumour could be felt. A parathyroid tumour ( $2.5 \times 1.5 \times 1.2$  cm.) was removed at operation. Pains in the bones disappeared a few days after operation. The urinary calcium excretion dropped to one-eighth of the pre-operative value. Three and a half years after the operation he still had no pains in the bones, and walked with a stick. The serum calcium at that time is reported as 13 to 14 mgrm. per 100 c.c., and no increase in density of the X-ray shadows of the bones had occurred.

### HYPERPARATHYROIDISM.

We now turn to the biochemical aspect of this problem. In 1926 Hannon, Shorr, McClellan, and Du Bois investigated the case of a man of 34 with the symptoms and signs of a generalized disease of the skeleton. Radiograms showed an extensive decrease in the density of the shadows of bones, and a large cyst in the right femur. There was a high blood calcium (serum Ca 15.3 mgrm. per 100 c.c.) and a low blood phosphorus (plasma inorganic P 1.4 mgrm. per 100 c.c.). In 1929 Richardson, Aub, and Bauer investigated the same case, and found that the urinary output of calcium and phosphorus on a low calcium diet was six to seven times greater than was found in normal individuals under identical conditions. They found that these changes in metabolism were equivalent to those of a normal individual receiving 100 units of parathormone daily. It was, therefore, reasonable to suppose that this patient was suffering from hyperparathyroidism. Exploration of the neck revealed no tumour, but this does not mean that no such tumour existed, for we now know that parathyroid tumours may be deeply situated behind the trachea or even in the mediastinum. It was the study of this case and the use of Aub's methods which led to the discovery of many cases of hyperparathyroidism in the United States and in this country. Meanwhile in Germany, Austria, Holland, and France the work of Mandl was producing the same effect.

### ABSTRACTS OF PUBLISHED CASES.

MANDL (Vienna, 1926).—Male, 38. Osteitis fibrosa of pelvis and both femora. Totally disabled by pain. Serum Ca 18.2 mgrm. Increased urinary excretion of

Ca. Parathyroid adenoma removed ( $2.5 \times 1.5 \times 1.2$  cm.). Urinary Ca excretion dropped to one-eighth the pre-operative value. Three and a half years later no pain. Walks with a stick. Has gained 16 kilos. Serum Ca 13 to 14 mgrm. No increase in density of X-ray shadows of bones.

LAMBIE (Edinburgh, 1927).—Female, 30. Generalized osteitis fibrosa. Serum Ca 17 mgrm., and inorganic P 2.7 mgrm. On a milk diet Ca output in urine normal, but considerable increase in faeces. No operation. Diagnosis confirmed at post-mortem, and adenoma of parathyroid found.

GOLD (Vienna, 1928).—Female, 54. Osteitis fibrosa generalisata (von Recklinghausen) with cysts in right humerus and in three ribs. Mal-union of fracture of left femur. Serum Ca 13.1 mgrm. Increased Ca excretion in urine. Parathyroid adenoma ( $2.5 \times 1.5$  cm.) removed. Serum Ca fell to 9.9 mgrm. one month after operation. Five months later much less pain, serum Ca 9.6 mgrm. Positive Ca balance. No increase in density of X-ray shadows of bones.

BARRENSCHEEN and GOLD (Vienna, 1928).—Sex and age not stated. Osteitis fibrosa cystica generalisata Recklinghausen. Pain in the bones. Serum Ca 14.2 mgrm. Slight increase of urinary Ca excretion. Removal of histologically normal parathyroid gland caused no clinical improvement. Two months later patient rather worse. Serum Ca 16.0 mgrm.

DUKEN (Jena, 1928). Case 1.—Female, 7. Osteodystrophia fibrosa. Pains in legs, tumour of frontal bone, protrusion of left eye. Tumour of 2 cm. diameter in left side of neck thought to be enlarged parathyroid. Serum Ca 14 mgrm., and inorganic P 5 mgrm. No operation. (Post-mortem report unsatisfactory and incomplete.)

Case 2.—Female, 14. Osteodystrophia fibrosa with late rickets. Ceased to grow at 12. Genu valgum. Hypotonicity of muscles. Polyuria. Fairly clear swelling in the neck (size and shape not stated). Serum Ca 20.8 mgrm. and inorganic P 3 mgrm. Resection of maxilla for tumour—histological examination showed osteitis fibrosa. No operation on neck.

BECK (Kiel, 1928).—Female, 41. Generalized osteitis fibrosa. Amputation of leg for supposed sarcoma. Three years later fell and fractured femur. True nature of condition then recognized clinically, confirmed by X rays, and proved histologically. Six years after the fracture Ca and P levels in blood raised (no figures given). Urinary Ca excretion three times the normal. Two parathyroid tumours, size of almond and coffee bean, removed. Three days after operation Ca and P values in blood normal, and Ca excretion in urine approximately normal. Tetany on fifth day, accompanied by a psychosis. Died of exhaustion on twentieth day.

RICHARDSON, AUB, and BAUER (Boston, 1929); HANNON, SHORR, McCLELLAN, and DU BOIS (New York, 1930).—Male, 30. Generalized disease of bones with extensive areas of decalcification and a large cyst in the right femur. Serum Ca 15.3 mgrm., and inorganic P 2.1 mgrm. Great increase in urinary Ca excretion. Exploratory operation on neck. No tumour found. Removal of two normal parathyroid glands. Two years after operation, considerable symptomatic improvement. Serum Ca 13.9 mgrm., and inorganic P 2.6 mgrm.

BARR, BULGER, and DIXON (St. Louis, 1929).—Female, 56. Osteitis fibrosa with benign giant-celled tumours of phalanx and ulna. Spontaneous fracture of clavicle. Hypotonia of muscles and joints. Increased frequency of micturition. Bilateral renal calculi. Serum Ca 16 mgrm., and inorganic P 1.4 mgrm. Great increase in urinary Ca excretion. Globular mass size of a small walnut felt embedded in left lobe of thyroid gland. Parathyroid adenoma removed (no measurements given). Severe tetany two days after operation. Calcium balance became positive. Swelling in superior maxilla became smaller.

WILDER (Rochester, Minn., 1929, 1931).—Female, 35. Generalized osteitis fibrosa with giant-celled tumours of inferior maxilla and femur. Six years, pain in hip ultimately affecting all bones. Serum Ca 13.2 mgrm., and inorganic P 1.9

mgram. Spherical mass palpable in neck: malignant adenoma of parathyroid gland removed ( $5 \times 3.5 \times 3$  cm.). Three days later tingling in fingers and toes, but no tetany; serum Ca 7.1 mgram. Great improvement, gave up crutches. Four months later serum Ca 8.3 mgram., and inorganic P 1.8 mgram., increased density of X-ray shadows of bones. Eighteen months after operation some pain in bones of hands, feet, and shoulders. Serum Ca and plasma P normal. X-ray shadows of bones still far from normal density, no cysts.

BOYD, MILGRAM, and STEARNS (Iowa City, 1929).—Male, 19. Generalized osteitis fibrosa. Progressive bowing of legs. Thirst and polyuria. Serum Ca 17.6 mgram., and inorganic P 2.2 mgram. Urinary Ca output three times the normal. Parathyroid adenoma removed ( $3.5 \times 2.5$  cm.). Chvostek sign appeared day after operation with serum Ca 12.3 mgram. Trembling of extremities with serum Ca 5.0 mgram., but no tetany. Polydipsia, polyuria, and pains in limbs disappeared. After ten days calcium balance positive. Three months after operation blood Ca and P normal; bones showed evidence of progressive repair.

SNAPPER (Amsterdam, 1929).—Male, 56. Osteitis fibrosa generalisata (von Recklinghausen) with cysts in scapula and metatarsal. Totally disabled by severe pains in bones. Spontaneous fracture of femur. Under observation four years, during which time density of X-ray shadows of bones decreased. Serum Ca 23.6 mgram., and inorganic P 2.1 mgram. Greatly increased Ca and P excretion in urine. Tumour palpable in neck: parathyroid adenoma removed ( $2.5 \times 1.5$  cm.). Day after operation pains ceased. Tremor, acute mania, Chvostek sign with serum Ca 6.6 mgram. Ca excretion in urine fell to normal. Four months later great clinical improvement, serum Ca 10.6 mgram. and inorganic P 3.3 mgram. Fracture healed, and after five months X-ray shadows of bones were more dense. Able to walk after eight months.

EGGERS (quoted by Mandl, 1929).—Female (age not stated). Generalized osteitis fibrosa. Serum Ca 14.6 mgram. Parathyroid tumour size of hazel nut removed. Serum Ca reduced to 5.7 mgram. Definite clinical improvement. Death by accident. Post-mortem confirmed diagnosis.

HUNTER (London, 1929, 1930, *Case 4*).—Female, 41. Generalized osteitis fibrosa. Three years, pain in back and left thigh. Four months, walked with sticks. Spontaneous fracture of neck of femur. Tenderness on pressure upon bones. No swelling palpable in neck. Radiograms showed greatly diminished density of shadows of bones. Serum Ca 16.7 mgram. and inorganic P 1.0 mgram. Plasma phosphatase 1.9 mgram. Ca output in urine six times the normal. Cystic parathyroid tumour removed ( $3.7 \times 3.0 \times 3.0$  cm.). Histology of tumour: hyperplasia of parathyroid gland. Histology of bone: osteitis fibrosa. Two days after operation serum Ca 9.1 mgram. Latent tetany only. On fourth day bones no longer tender to pressure. Ca output in urine fell approximately to normal. Eight months after operation she walked in a caliper splint, and seven months later unaided. No further pains in bones. Seventeen months after operation she had gained 2 st. 6 lb. in weight. Serum Ca and inorganic P remained normal. Plasma phosphatase remained high. In controlled radiograms density of bone shadows had increased slightly, but was not nearly normal.

BARR and BULGER (St. Louis, 1930, *Case 2*).—Male, 38. Multiple giant-celled tumours of bone, localized to upper and lower jaws. No obvious decalcification or osteomalacia of other bones. Hypercalcaemia up to 16.7 mgram. Inorganic P as low as 1.2 mgram. Negative calcium balance. Tumour palpable just above inner end of left clavicle, especially when swallowing. Parathyroid tumour removed; twice as large as was expected from palpation. Part of it was re-implanted in the rectus sheath. In twenty hours the serum Ca dropped to 10.6 mgram. and reached its minimum ten days after operation at a level of 8.3 mgram. Urinary output of Ca decreased. Ca balance became positive. No tetany occurred. Two months after operation quite well. Weight increased. Serum Ca 10.5 mgram. Inorganic P 2.9 mgram. X-ray examination showed no progress of bone disease. The case had been treated earlier with X rays because of a diagnosis of malignant tumours of bone.



ASK-UPMARK (Lund, 1930).—Male, 46. Osteitis fibrosa generalisata (Engel-Recklinghausen). Twelve months, pain in feet, knees, and hips, later becoming very severe. Spontaneous fractures in femur and humeri. Great wasting. Radiograms of bones showed general decalcification. Serum Ca 10.0 mgrm. At autopsy only one parathyroid gland identified, this being more than four times normal size.

BALL (Rochester, Minn., 1930).—Male, 50. Generalized osteitis fibrosa. Seven years, pain in dorsa of feet, later affecting knees, lumbar spine, right hand, and wrists. Four attacks of ureteric colic with passage of gravel on one occasion. Spontaneous fracture of 1st lumbar vertebra. Epigastric pain, anorexia, and mild nausea. Kyphosis. Nodule palpable in lower pole of right lobe of thyroid gland. Hypotonia of muscles. Radiograms of bones showed multiple areas of absorption. Serum Ca 15.4 mgrm. Inorganic P 1.7 mgrm. Plasma phosphatase increased. Negative calcium balance. Double resection of thyroid gland. Cystic colloid and foetal adenoma. No parathyroid tissue found. After operation serum Ca remained high and inorganic P low. Excretion of calcium remained above normal level.

COMPERE (Chicago, 1930).—Female, 59. Generalized osteoporosis with no sign of bone cysts or tumours. Five years, pain in bones of legs, particularly feet. Two and a half years, bowing of both legs and general weakness. Hypotonicity of muscles. Spherical nodule, 2 cm. in diameter, palpable in lower pole of right lobe of thyroid gland. Serum Ca 12.6 mgrm. Inorganic P 3.7 mgrm. Increased urinary output of calcium. At operation nodule when excised proved to be thyroid adenoma. Parathyroid adenoma (1.0 × 1.8 cm.) removed from behind lower pole of left lobe of the thyroid gland. Normal left upper parathyroid body also removed. Developed tinglings in fingers and numbness in hands. No tetany. Serum Ca fell to 6.9 mgrm. after two days, but after two months was 11.1 mgrm. Urinary Ca output reduced to one-fifth of pre-operative level. No change in radiograms of bones after five months. General symptomatic improvement with gain in weight.

PEMBERTON and GEDDIE (Rochester, Minn., 1930).—Female, 14. Generalized osteitis fibrosa. Sixteen months, progressive weakness, loss of weight, vomiting, polydipsia, polyuria. Hypotonia of muscles. Radiograms showed diffuse decalcification of bones but no loss of normal structure. Serum Ca 16.3 mgrm. Inorganic P 2.5 mgrm. Negative Ca balance. No tumour palpable in neck. Adenoma of parathyroid gland removed (1.5 × 1.3 × 1.3 cm.). After operation gastro-intestinal and other symptoms rapidly improved. Serum Ca 7.6 mgrm. Inorganic P 3.9 mgrm.

LÉRI, LAYANI, LIÈVRE, and WEILL (Paris, 1930).—Male, 31. Osteitis fibrosa of von Recklinghausen. Five years, pains in right leg. Four years, attack of renal colic. Three years, pain on walking, swellings in tibiae and left ulna. Two years, pain in left hip, spontaneous fracture of left femur. Swellings and tender areas in bones. Radiograms showed diffuse decalcification of skeleton with cyst formation. Serum Ca 19.8 mgrm. Histological section of ulna showed osteitis fibrosa with giant cells. Later, other spontaneous fractures causing severe skeletal deformity. Further attack of renal colic. Parathyroid tumour size of small bean removed. Histology: adenomatous hyperplasia. Tetany on tenth day. Great improvement. Sixteen months after operation: gained weight, walked with sticks, serum Ca 12.7 mgrm. Increased density of X-ray shadows of bones, with decrease in size of cystic areas.

FRASER (London, 1930). Case 1.—Female, 42. Generalized osteitis fibrosa with cysts of bone. Ten years, pain in bones. Spontaneous fractures of both humeri. Many cysts in long bones everted. Radiograms showed decreased density of shadows of bones with cyst formation. No renal calculi seen. Serum Ca 14.6 mgrm. Parathyroid adenoma removed. Serum Ca day after operation, 5.9 mgrm. Pains in bones disappeared. Able to walk a mile. Seven months later returned with pains in arms and thighs; serum Ca 8.2 mgrm.

Case 2.—Female, 26. Diffuse osteitis fibrosa. Three years, pain in bones. Admitted with spontaneous fracture of left femur. Radiograms showed generalized changes in bones; small renal calculi in both kidneys. Serum Ca 16.4 mgrm.

Cystic adenoma of parathyroid gland removed. Serum Ca day after operation, 7.5 mgrm. Fracture united. Three months after operation: pains in bones still present; unable to walk without assistance; no increase in density of X-ray shadows of bones: serum Ca 9.6 mgrm.

*Case 3.*—Female, 23. Fibrocystic disease of bones. History of fall when aged 14, followed three years later by swellings of terminal phalanges of fingers and of wrists. Cysts enretted: sections showed osteitis fibrosa with cyst formation. Marked deformity of hands, long bones, pelvis, and skull. Large bilateral renal calculi. Serum Ca varied between 12.0 and 15.2 mgrm. Parathyroid tumour removed. Day following operation, serum Ca 7.3 mgrm.

WENDEL (Magdeburg, 1930, *Case 2*).—Female, 17. Generalized osteitis fibrosa. Two years, had to give up sports because of sacral backache. Twelve months, fracture of left femur, shortly followed by fracture of right femur. Radiograms showed multiple foci of osteitis fibrosa, especially in both femora, humeri, and spine. Serum Ca varied between 15 and 18.9 mgrm. Head of left fibula explored; histological section showed benign giant-celled tumour. At operation left lower and right upper parathyroid glands noticeably enlarged (no measurements given). Left lower parathyroid gland removed and found histologically normal. After operation serum Ca remained high.

ROSENBACH and DISQUÉ (Potsdam, 1930).—Female, 24. Generalized osteitis fibrosa. Severe thirst and polyuria suggesting diabetes insipidus. Pains in head, back, and limbs, loss of weight, muscular weakness, vomiting, loss of appetite. Spontaneous fractures of left femur and right humerus. Radiograms showed severe generalized osteitis fibrosa. (Blood chemistry not recorded.) Great increase in urinary Ca excretion. Soft grey tumour of right inferior parathyroid gland removed (no measurements given). Histology: adenoma or hyperplasia. After operation vomiting abruptly ceased and appetite returned. Oliguria. Urinary Ca output fell to one-fifth of pre-operative level. Walked with aid of sticks.

HURST and COSIN (London, 1931).—Male, 17. Generalized osteitis fibrosa. Three years, genu valgum. Spontaneous fracture of left femur. Large swellings in each thigh. Two years, noticed small tumour in neck. Severe deformities of bones with tenderness on pressure. Firm oval tumour felt deeply situated in neck on left side of trachea. Excess of dark hair on pubes, abdomen, and back. Radiograms: greatly diminished density of all bones; trabeculated cysts in both femora with pathological fractures. Bilateral renal calculi. Serum Ca 16.5 mgrm. Inorganic P 2.1 mgrm. Plasma phosphatase increased. Urinary calcium output increased. Cystic adenoma ( $2.7 \times 2.2 \times 2.0$  cm.) of parathyroid gland removed from behind upper pole of left lobe of thyroid gland. Tingling of fingers day after operation. On third day Chvostek and Trousseau signs positive. Fifth day, serum Ca 7.8 mgrm., inorganic P 3.9 mgrm.

QUICK and HUNSBERGER (Philadelphia, 1931).—Male, 25. Five years, polydipsia, polyuria. Pains in back, arms, and legs. Persistent nausea. Four years, spontaneous fracture of femur, followed by other fractures. Second end of left radius explored; histological section showed . . . . . Three years, deformities progressed, multiple swellings appeared in various bones. Hæmaturia. No renal colic. One year, extreme deformity of all bones except those of head, hands, and feet. Diminution of 25 in. in total height. Radiograms showed marked decalcification of all bones, with innumerable fractures, and evidence of tumours and cysts. Discrete dense shadows in region of left kidney. Serum Ca 15.7 mgrm., and inorganic P 2.7 mgrm. Plasma phosphatase increased from four- to five-fold. Fullness in left side of neck suggestive of a tumour. At operation parathyroid tumour ( $1.7 \times 1.5 \times 1.1$  cm.) removed from deep surface of left lobe of thyroid gland. Histological picture: hyperplasia. In one week serum Ca fell to 11.5 mgrm., but in three months had risen again to 15 mgrm. At second operation, three months after first, further portion of tumour ( $3.5 \times 2.0 \times 1.5$  cm.) removed. Twenty-four hours later, serum Ca 8.9 mgrm. Tetany on fourth day relieved by calcium gluconate and calcium chloride given intravenously. No tetany after

thirteenth day. Estimation of Ca balance showed an increased positive balance compared to the figure obtained before operation. Radiograms taken one month after second operation showed increased density of shadows of bones.

SNAPPER and BOEVE (Rotterdam, 1931, *Case 2*).—Female, 35. Three years, swelling of right heel, radiograms showed cyst in calcaneus. Two years, swelling in right tibia explored; histological section showed giant-celled tumour. Four months, pain in right thigh, spontaneous fracture of right femur which remained un-united. Radiograms showed poor bone shadows with cysts in ilium and femora. No tumour felt in neck. Serum Ca 14.7 mgrm. At operation adenoma of parathyroid gland size of almond removed. No tetany, pains improved. Two months later serum Ca 12.1 mgrm., fresh cyst-like areas in radiograms of bones. Neck re-explored but no further tumour found. Patient treated with vigantol (irradiated ergosterol); great improvement, both clinical and radiological, although serum Ca 13.8 mgrm. and inorganic P 0.84 mgrm.

HUNTER (London, 1931).—Female, 37. *Case 2* of present paper.

### METHODS USED IN STUDY OF AUTHOR'S CASES.

**Methods of the Metabolism Ward.**—In our experience it has been impossible in the general hospital ward to attain accuracy of method which in any way approaches that of the laboratory. Our investigations have been carried out in a special metabolism ward under the care of an experienced Sister-Dietitian, and it is upon the painstaking and intelligent efforts of such a person that the accuracy of the work depends.

In the study of calcium metabolism it is necessary to know not only the level of calcium and phosphorus in the blood, but also the calcium and phosphorus balance; for just as the level of a river does not indicate the direction in which it flows, so the levels of the figures in the blood give us, in themselves, no indication whether calcium is flowing into excretory channels or into the tissues.

In our studies of endogenous calcium metabolism we assume a definite responsibility to the patient for the proper treatment of the disease under consideration, and we see that this has precedence over any investigations. The patient is given a diet containing only 100 mgrm. of calcium a day, but adequate in all other respects. This is necessary in order to reduce to a minimum the amount of unabsorbed calcium in the faeces. During the first few days the patient is allowed to choose whatever articles of diet seem most attractive from the limited list of foods low in calcium (Sherman, 1926; Simmonds, 1931). Emphasis is placed upon the fact that once a diet is chosen it cannot be changed during the period of study. Fluid and sodium chloride intake are kept constant, and distilled water is used in the preparation of all food and drinks. Sufficient sodium bicarbonate is given by mouth to make the reaction of the twenty-four-hour mixed urine neutral to phenol-sulphonaphthalein (pH 7.3). The faeces are divided into three-day periods by the oral administration of 0.3 gm. of carmine alum lake every third day. Since constipation usually occurs on a low calcium diet we frequently employ liquid paraffin as a laxative. In some cases, where carmine does not appear in the stools within twenty-four hours, we use a simple enema made with distilled water. The urine is obtained in twenty-four-hour specimens.

Since inorganic salt excretion reaches a constant level only very slowly,

we endeavour to change our conditions of study not more than once in twelve days.

Preparatory to operation for removal of a parathyroid tumour the patient is kept on an unweighed high calcium diet. This includes four pints of milk daily, together with ice-cream, cheese, and eggs.

**Laboratory Methods.**—In estimating the blood-serum calcium the Kramer-Tisdall method as modified by Clark and Collip (1925) gives results with an average error of 2 per cent and a maximum error of 5 per cent. The normal figure by this method varies between 9.0 and 11.0 mgrm. per 100 c.c.

The distribution of phosphorus changes when blood is allowed to stand. Once the corpuscles have been laked, the liberated organic esters are very rapidly hydrolysed by the plasma phosphatase, and the concentration of inorganic phosphates rises rapidly. Samples of blood for estimation of the inorganic phosphorus must therefore be taken into oxalate, and any hæmolyzed specimens must be rejected. With such precautions, using the method of Briggs (1924), the experimental error should not exceed 2 per cent.

In health the level of inorganic phosphate in the blood plasma varies between 2.5 and 3.5 mgrm. phosphorus per 100 c.c. In children, where ossification is more active, readings up to 5 mgrm. per 100 c.c. are found.

In 1929 Kay published a method for estimating the activity of the phosphatase in the plasma. He expresses this enzyme activity in terms of the number of milligrams of phosphorus liberated as inorganic phosphate from excess of sodium  $\beta$ -glycerophosphate in forty-eight hours at 38° C. and at pH 7.6 by 1 c.c. of plasma. Under these conditions the average value for normal plasma is 0.15 mgrm. In young infants the figure is higher.

The calcium estimations of both food and excreta were carried out by the method of McCrudden (1910).

**Radiographic Methods.**—In estimating the density of the radiographic shadows of bones it is found necessary to use a controlled method. A control subject is chosen of the same sex, age, weight, height, and build as the patient. The corresponding limbs of patient and control are placed side by side and exposed simultaneously on the same negative. Where a series of radiograms is taken in one patient the same control subject is used each time.

**Histological Methods.**—Wherever possible histological investigations of portions of bone and of parathyroid tumours have been carried out. The methods are indicated by Professor H. M. Turnbull in the reports on the corresponding cases.

## CASE REPORTS.

*Case 1.*—Hyperparathyroidism. Generalized osteitis fibrosa. Renal calculi. (By the kindness of Dr. N. Hill and Dr. H. C. Lucey).

E. G., married woman, aged 49.

**HISTORY.**—Eight years, back began to bend. Shoulders became rounded. Gradual diminution of total height. Four years, multiple spontaneous fractures giving rise to deformities of limbs. Became bed-ridden. Admitted to Metropolitan Hospital, London, with spontaneous fracture of left humerus.

**Past History.**—Had pneumonia twice. Four children. Three alive and well. One died of tuberculosis.

**ON EXAMINATION.**—Height, 4 ft. 9 in. Intelligence normal. Lies in bed

totally disabled, greatly deformed, breathless, and very wasted. Calvaria large. No tumour felt in neck. Angular curve in spine at level of 5th thoracic vertebra. Sternum and clavicles arched forwards. Collapse of ribs, causing the lateral diameter of the thorax to be greatly diminished and the antero-posterior diameter increased. Gross deformity of both upper limbs from mal-union of fractures. Signs of fracture of left humerus. Abdomen protuberant, subcostal margins approximated to iliac crests. Legs crossed scissor fashion owing to mal-union of fracture of left femur.

**RADIOGRAMS.**—Poverty of shadows of all bones examined, loss of detail and stippling. Atrophic changes, pale cyst-like spaces, and old fractures in bones of limbs. Kyphosis, collapse of ribs, and tri-radiate pelvis. Calvaria greatly thickened, blurred, and coarsely mottled. Large calculi in right kidney.

**PROGRESS.**—Progressive emaciation and exhaustion. Serum calcium 13.3 mgrm. per 100 c.c. Died following further spontaneous fractures.

**POST-MORTEM (Dr. H. C. Lucey).**—Bronchopneumonia. Gross changes in bones and parathyroid glands (*see below*). Three large irregular calculi in right kidney, with hydronephrosis and dilated ureter. No calculi in left kidney. Heart, liver, and spleen wasted. Larynx, trachea, œsophagus, suprarenals, pancreas, uterus, ovaries, and pituitary normal. No macroscopic evidence of metastatic calcification found in any organ.

**COMPOSITION OF RENAL CALCULUS.**—A portion of calculus taken for chemical analysis was friable and of a uniform white colour and consistency throughout its whole substance.

Chemical composition :—

Ca ..	..	24.4 per cent	CO <sub>2</sub> ..	..	0.7 per cent
P ..	..	8.4 ..	Oxalic acid ..	..	0.85 ..

The calculus therefore consists principally of calcium phosphate, but there is more calcium than can be entirely accounted for by the acids. A comparison with the composition of bone is obviously of great interest.

Composition of bone :—

Ca ..	..	20.0 per cent	CO <sub>2</sub> ..	..	3.25 per cent
P ..	..	9.0 ..			

### Pathological Report.

(PROFESSOR H. M. TURNBULL.)

Tissues sent by Dr. H. C. Lucey and Dr. N. Hill. (P.M. Appendix 496/1929.)

#### 1. Bones.—

##### MACROSCOPIC.—

1. *Calvaria.*—This and the other specimens had been preserved by the Kaiserling process. The specimen was the posterior part of the roof of the skull removed by a coronal cut close to the fronto-parietal suture and a horizontal cut 1.5 cm. below the external occipital protuberance. The bone was greatly thickened and weighed 503.3 grm. It was 0.7 cm. thick at the junction of the two cuts, and rapidly thickened to 1.5 cm. upon the top of the skull and to 2 cm. in the mid-line below the occipital protuberance. The sawn surfaces were finely porous under a hand-lens, and red-purple. The grooves for the meningeal arteries were very deep; the sutures were completely obliterated.

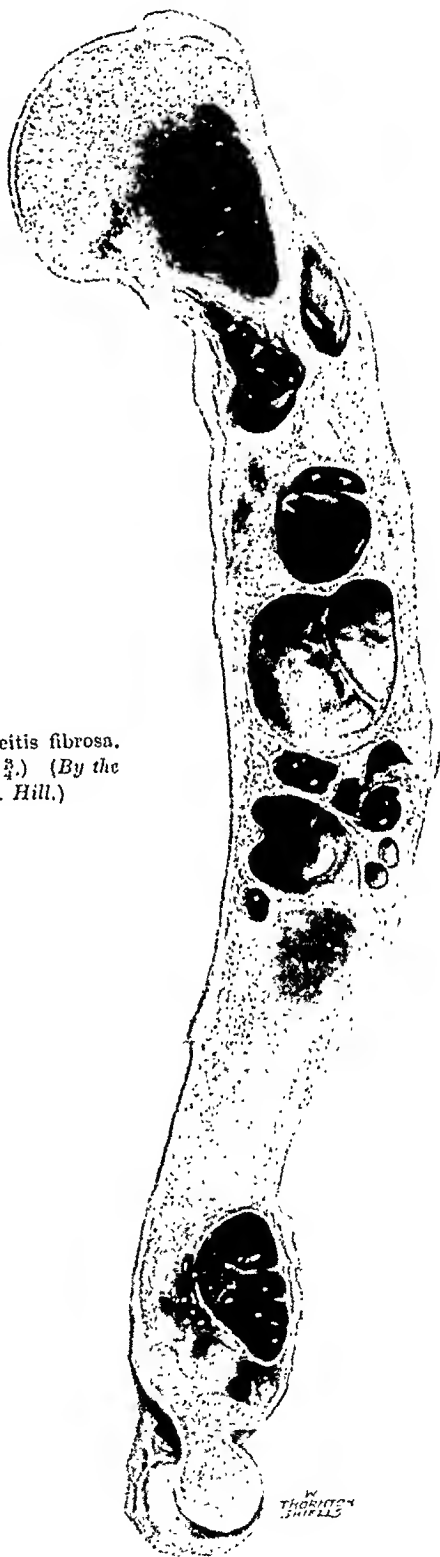
2. *Right Humerus (Fig. 175).*—The bone was greatly deformed, and measured only 25 cm. in a straight line from top to bottom. The shaft showed a conspicuous curve forwards after a sharp kink at the surgical neck and also a sinuous curve with the upper convexity outwards and the lower inwards. A fusiform expansion (3.3 cm. diam.) in the upper 9 cm. of the shaft was followed by a narrow portion (2 cm. diam.) for 3.5 cm., and this again by an expansion (3 cm. diam.) above the capitellum and trochlea. The lower expansion formed upon the anterior surface an oval, dark swelling (5 × 3.5 cm.) with an apparently membranous wall. The shaft was covered by an adherent layer of periosteum, tendon, and muscle. On the cut

surfaces revealed by sagittal section the tuberosities and the head showed a few small areas of soft yellow fatty marrow enclosed in a grey gelatinous tissue, which felt gritty and showed in places under a hand-lens a close, spongy bone. In the lower part of the tuberosities and in the surgical neck was an area of red marrow, free from bone. In the surgical neck this red marrow was bounded on each side by a layer (3 mm. deep) of grey gelatinous tissue that contained a delicate porous bone of slightly wider mesh than in the head. The red marrow extended as a wedge into the upper expansion of the shaft. The rest of the upper expansion contained five large and many small cysts of round or irregular shape. The cysts were often crossed by membranous trabeculae, and were either empty or contained a deep-red jelly. The lining of the empty cysts was smooth and slaty grey. The larger cysts had a thick (2 mm. or more) wall of elastic grey tissue. Between the cysts was a little grey, gelatinous, tough, granular tissue mottled with red. Grey tissue containing spicules of bone, similar to that forming the

FIG. 175.—Case 1. Generalized osteitis fibrosa. Sagittal section of right humerus. ( $\times \frac{2}{3}$ .) (By the kindness of Dr. H. C. Lucey and Dr. N. Hill.)

corticalis of the neck, lay beneath the periosteum over the cysts. It usually formed a mere shell, but at one spot on the anterior surface was a small patch 5 mm. thick and at another a longer segment 5 mm. thick. Beneath the latter patch was an area of yellow, fatty marrow. In the lower end of the upper expansion, beneath the cysts, the medulla was filled with soft red-brown tissue. In the narrow portion of the shaft the medulla contained yellow, fatty marrow. The corticalis here and outside the red-brown tissue above consisted, as in the neck, of grey gelatinous tissue containing a delicate spongy bone. Where fairly cut it was 4 mm. thick. In the lower expansion cysts similar to those in the upper were covered anteriorly by a gritty membrane and posteriorly by about 4 mm. of the grey gelatinous tissue containing spicules of bone. The wall between the fossae above the trochlea consisted of a similar grey, gritty tissue; the trochlea contained a harder, finely porous bone and fatty marrow.

**Microscopic.**—Portions of the skull and humerus were decalcified in Müller's solution followed by nitric acid, and were stained by Schmorl's thionin methods, etc.



1. *Calvaria, at Summit of Coronal Cut.*—The bone is limited externally and internally by an interrupted line of slender trabeculae. Between these lines trabeculae of very irregular shape, either lying free or anastomosing, form a spongiosa of close mesh. The trabeculae consist of a non-lamellar or woven bone. Part of this is typical coarse-fibred bone, in which the bundles of fibrils interlace at obtuse angles, the cell-spaces are closely packed, large and of irregular shape, and the canaliculi are scanty and short. Part is a bone in which the bundles of fibrils interlace at more acute angles and in places form an imperfect lamellation, whilst the cell-spaces and their canaliculi approach in number, arrangement, and shape those of perfect lamellar bone. Those two types of woven bone may be conveniently called *coarse-fibred bone* and *intermediate bone*. Where both occur in one system the coarse-fibred is central and merges into the intermediate, which may become imperfectly lamellar in its periphery. The imperfectly lamellar bone resembles that laid upon cartilaginous trabeculae in infants; it is an immature lamellar bone. There is no perfect lamellar bone, such as is formed in the Haversian systems of adults. The larger trabeculae usually show three or four systems in any field under a  $\frac{1}{8}$ -in. objective. A few trabeculae contain small vascular canals, but there are no Haversian systems. There are numerous focal areas of resorption by many large osteoclasts. Active apposition is also focal. The osteoid zones are not abnormally deep. The marrow is fibrous throughout. The fibrosis is very uniform, and consists of numerous delicate collagenous fibrils and relatively few fibrocytes with narrow spindle nuclei. The vessels are engorged but are narrow. Near the outer surface is an oval cyst ( $5 \times 2.5$  mm.), which has a smooth margin bounded by a narrow zone of denser fibrosis.

2. *Head and Under Surface of Anatomical Neck of Humerus.*—There is a narrow layer of bone beneath the articular cartilage, and a very slender, interrupted, strip of bone beneath the periosteum of the neck. These layers of bone are bounded by a zone of fibrous marrow, except in a very few places, where they are in contact with adipose marrow. Within the head and neck is a remarkably small number of small, irregular, and slender trabeculae. One or two of these are bounded by adipose marrow in part of their course. A few others are surrounded by a narrow zone of fibrous marrow. All the remainder are grouped within broad strands of fibrous marrow which form a net enclosing large areas of adipose marrow. The adipose marrow is in excess of the fibrous. In the few places in which the bone is in contact with adipose marrow it is almost invariably undergoing neither resorption nor apposition. Elsewhere active resorption is very great. Osteoclasts are very numerous both upon and close to the surfaces of trabeculae, which have a very gnawed appearance owing to the number of Howship's lacunae. Active apposition is shown by numerous large osteoblasts upon osteoid zones. It is greatly overshadowed by active resorption. Where the osteoid zones are cut at right angles to their surface, they are not deeper than in the long bones of healthy infants. The fibrous marrow is slightly more cellular than in the skull. With the exception of one or two small fragments of lamellar bone the bone is woven and mostly of the intermediate type.

3. *Surgical Neck.*—The corticalis is replaced by a sharply defined layer of fibrous marrow within which lie scattered a few small trabeculae of woven bone. Active resorption by osteoclasts is again very great. Active apposition is present but is relatively slight. The medulla consists of adipose tissue which is injected throughout and contains numerous foci of hæmatogenous activity.

4. *Shaft between Upper and Lower Expansions (Fig. 176).*—The longitudinal section includes the whole thickness of the bone. The corticalis on each side is again replaced by a layer of fibrous marrow, which is of even depth and has a straight inner margin. Under the periosteum on each side is an interrupted, narrow strip of bone. This is lamellar on the outer side but woven on the inner. Beneath this strip the fibrous marrow contains trabeculae which are in general slightly more numerous and larger than in the surgical neck. They consist of woven bone, which for the most part is of the intermediate type and frequently becomes lamellar towards the free surfaces. One or two Haversian systems are being formed within

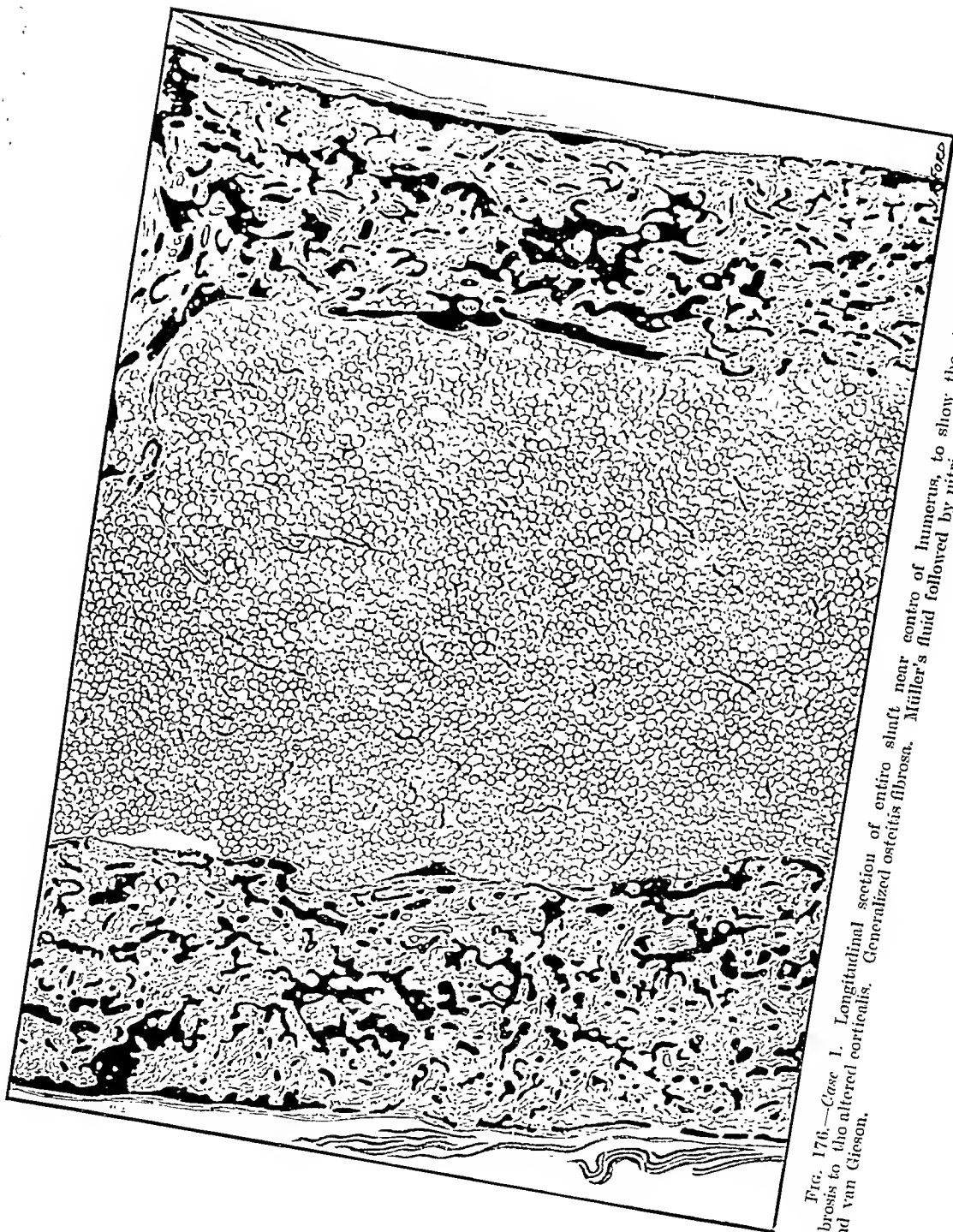


FIG. 176.—Case 1. Longitudinal section of entire shaft near centre of humerus, to show the strict limitation of the fibrosis to the altered corticis. Generalized osteitis fibrosa. Müller's fluid followed by nitric acid; Weigert's iron-haematoxylin



the largest trabeculae. The trabeculae are composed of fewer separate systems than in the skull. In many places there are numerous osteoclasts in Howship's lacunae and in the adjacent marrow. There are also many osteoid zones covered by a layer of closely packed, large osteoblasts. The osteoid zones are of about the same depth as in infants. The fibrous marrow is very similar to that in the head of the humerus, but the capillaries are slightly widened, there are a few hæmorrhages, and there are many groups of cells loaded with iron-pigment. Further, there are small focal areas in which the marrow is not fibrotic and contains hæmatogenous marrow. The medulla contains adipose tissue.

**Remarks.**—The most conspicuous feature in the bones examined is resorption. Resorption was very active at the time of death, especially in the cortex of the humerus. Past resorption is obvious in view of the replacement of the compacta of the skull and humerus by small isolated trabeculae. In the humerus extremely little bone remains. All perfect, adult lamellar bone has been removed from the skull and humerus, except possibly some small fragments beneath the periosteum of the humerus. The bone that is now present is almost all woven bone which is partly typical coarse-fibred bone and partly a woven bone that has been described above and called 'intermediate bone'. The coarse-fibred bone merges into the intermediate bone, and the latter merges into a lamellar bone which is most perfect in the corticalis of the narrow portion of the shaft of the humerus. Coarse-fibred and intermediate bone are found in the fundamental systems of the long bones of adults. Since, therefore, resorption usually commences in Haversian systems and extends to fundamental systems, portions of such bone are likely to remain longest. The amount and distribution of the woven bone in this case, however, make it impossible for it all to be remnants of fundamental systems. Further, much is obviously of recent formation. There can be little doubt that most of the bone present, probably all in the skull, represents bone which has been formed during the disease. It follows that resorption has cleared away almost all, if not all, the normal bone, and that this great resorption has been associated with apposition. That the two processes have continued side by side for a considerable time is shown by the mosaic of systems in the trabeculae. The number of systems is small in the small trabeculae of the humerus, but is moderately great in the larger trabeculae of the skull. At the time of death active apposition was taking place throughout the bones examined, but is overshadowed by the active resorption. The number and size of the osteoblasts upon the osteoid zones point to a rapid apposition. The depth of the osteoid zones is not greater than in healthy infants, so that in view of the rapidity of apposition there is no evidence of osteomalacia. When sections such as these are examined it is not difficult to understand how bones formed by trabeculae isolated in a fibrous matrix can bend without any softening of the trabeculae.

A fibrous marrow is present throughout the calvaria, throughout the corticalis of the shaft of the humerus, and in part of the spongiosa of its head and neck. The distribution of the fibrous tissue is closely connected with the osseous portions of the bones. In the shaft of the humerus it replaces the original corticalis and is sharply limited from the adipose or hæmatogenous marrow of the medulla (*Fig. 176*). In the head and neck of the humerus it forms strands enclosing groups of bony trabeculae or forms a zone round

single trabeculae. A few trabeculae are in part of their course in direct contact with adipose marrow on one or both sides. At such spots active resorption and apposition are very rare, but do occur. These appearances, which I have seen in other cases, suggest that the fibrosis of the marrow is merely an expression of exceptional activity in the osteogenetic marrow.

The cysts in the humerus were not examined microscopically, but a small cyst was accidentally found in the skull. Its structure gives no indication of its mode of formation.—H. M. T.

## 2. Parathyroid Bodies.—

**MACROSCOPIC.**—The specimen was a thyroid gland (*Fig. 177*) from which had been sliced off the posterior parts of the left lateral lobe. The cut surface of the thyroid gland was dark and glistening. In a concavity in the upper pole of the left lobe was the cross-section of a sharply defined yellowish-white sphere 1.1 cm. in diameter. This was covered externally by a transparent capsule, which swept in as a narrow grey line between it and the thyroid tissue. Projecting into the lower pole of the right lobe from the posterior border was a yellowish-white mass, 2.8 cm. high, 1.8 cm. from before back, and about 2.5 cm. from side to side. The outer surface was nodular and covered by a transparent capsule. The cut surface showed a central, granular, gritty, yellow area, from which one or two calcareous spicules projected. Round this was a zone of smooth, pale yellow tissue separated into rounded lobules of about 3 mm. in diameter.



*FIG. 177.*—Case 1. Generalized osteitis fibrosa. Thyroid gland showing in section smaller tumour of left superior parathyroid body and larger tumour of right inferior parathyroid body. (Actual size.) (By the kindness of Dr. H. C. Lucey and Dr. N. Hill.)

**Microscopic.**—The *left upper parathyroid body* is surrounded by a capsule of dense collagenous fibrous tissue and elastic fibres, which shows a few calcareous plaques in its inner aspect. Within this it is divided into small acini by a net of capillaries, walled by a sheet of collagen. In three small areas the collagen is greatly swollen and hyaline, whilst the capillary lumina are obliterated, so that stout hyaline nets are formed enclosing small remnants of acini. Throughout much the greater part of the gland the acini contain polygonal cells with a deeply oxyphil, eosinophil, cytoplasm. This cytoplasm is almost always vacuolated, but the vacuoles are usually very small, so that the cells typically have a solid, granular appearance, recalling hepatic cells. The cells measure in mean diameter 7 to 11.5  $\mu$ . Their nuclei are round or slightly oval. They usually measure 4 to 5  $\mu$ , but variation in the size of the nuclei is conspicuous; many measure up to 7  $\mu$ , and there are a very few of 9.5  $\mu$ . Most are very deeply stained. They show a stout membrane, a widely meshed net of chromatin, large rounded nodes, and occasionally, one or more nucleoli. In sharp contrast to the main part of the gland are a few scattered groups of acini which contain smaller, greatly vacuolated cells. These cells measure from 6 to 9  $\mu$ . Their nuclei are usually 4  $\mu$ , rarely as large as 6  $\mu$ ; the larger examples are less deeply stained. They show a basophil reticular cytoplasm and a basophil cell-membrane (pyronin-methylgreen). Occasionally a faintly stained oxyphil substance can be seen in the meshes of the reticulum. Most of the cells are very dropsical, showing little or no cytoplasm except the cell-membrane. Some of these acini contain a few of the deeply oxyphil cells also.

In the adjacent thyroid gland most of the acini are large and filled with colloid,

whilst the epithelial cells are sharply defined and flattened. In other acini there is little or no colloid, and the cells are ill-defined.

The *right lower parathyroid body* is divided into large rounded and oval lobules by stout septa, which consist of collagen and elastic fibres and are frequently bounded internally by a sheet of elastic fibres. The peripheral lobules project into the surrounding areolar tissue, being frequently separated to a considerable extent by wedges of areolar tissue containing large vessels, and their free convexities are encapsulated by continuations of the septa. One large and several small lobules lie apparently free in the areolar tissue at a considerable distance from the main mass. Other small lobules appear to be isolated within septa. The capsules of the isolated lobules are nowhere lined with endothelium. The septa contain veins and a few arteries. The lobules are more or less completely subdivided into acini by the walls of numerous branching veins and capillaries (Laidlaw's and van Gieson's stains). The acini are usually of large size and are frequently long and narrow. In one lobule there are large areas in which the collagen of the veins and capillaries is greatly swollen and hyaline, and forms hyaline nets like those seen in the left body. Most of this hyaline tissue is calcified. In the centres of the areas the remnants of acini within the calcified net are frequently necrosed and often are calcified. The septum bounding this lobule, and some adjacent septa, are also hyaline and extensively calcified. In this body deeply oxyphil cells are very scanty. Usually they are scattered singly throughout the gland, but sometimes they form small groups. They are of similar size to those in the left. Basophil cells greatly predominate. They measure from 4.5 to 12  $\mu$ . Most are between 9 and 12  $\mu$  and are dropsical, the cytoplasm being reduced largely or entirely to a cell-membrane. The nuclei are usually 4  $\mu$  in mean diameter, but there are a few giant oval nuclei up to 10.5  $\mu$ . Most of the nuclei are pale. In places these dropsical basophil cells are of long, narrow columnar shape, e.g.,  $20 \times 3 \mu$ . They are then arranged sometimes in a palisade between two vascular septa, sometimes as the sharply defined wall of a cleft that contains a few free rounded cells. Cubical basophil cells occasionally surround a small sphere of oxyphil coagulum.

In a small piece of thyroid gland the colloid is less deeply stained than on the left, and the cells are not flattened.

**Remarks.**—In order to compare the cytology of this and the subsequent enlargements with that of normal parathyroid bodies some account of the latter is necessary. The measurements given are mean diameters in paraffin sections of a series of glands fixed in formaldehyde.

#### *The normal parathyroid glands.*

Welsh (1898) divided the cells of the normal gland into oxyphil\* cells and principal cells; he subdivided the latter into four types according to their form and arrangement. Erdheim (1903) described two kinds of oxyphil cells: the large pale and the small dark. According to Erdheim (1901) oxyphil cells occur first in the tenth year, and are present in variable number ever after. My own series agrees with this, but only one year, the fifth, is represented between the second and the thirteenth. There is no difficulty in distinguishing these three types of cell: the dark oxyphil, the pale oxyphil, and the principal.

The *dark oxyphil cells* are in general the rarest. They are frequently absent when the pale type is present, and it is exceptional to find them forming large groups. They are usually found in small number, both among the principal cells and in contact with pale oxyphil cells. The latter is their

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\* Welsh actually wrote "oxyphile."

favourite position. When present in groups of pale oxyphil cells they recall crescents in mucous glands. They measure from 7 to 12  $\mu$  in mean diameter, usually being about 9.5  $\mu$ . The cytoplasm looks dense and granular. Under high magnifications it shows scattered vesicles of different sizes; I have not detected true granules in fixation with formaldehyde, Zenker, or alcohol. It is stained deep red or purplish red by hæmatoxylin and eosin, and dark green by pyronin-methylgreen. No net formed by cell-membranes is seen between adjacent cells, but in sections stained with pyronin-methylgreen a pink membrane sometimes bounds the cytoplasm of an occasional cell. The nucleus measures 3.5 to 5  $\mu$ . Except when of the largest size, it is usually deeply stained but shows a clear structure.

The *pale oxyphil cells* (Fig. 190) usually form a variable number of nodules that are conspicuous under low powers; with higher magnifications others are found singly or in small groups. The cells measure from 9.5 to 17.5  $\mu$ , but are usually from 12 to 15  $\mu$ . The cytoplasm is very characteristic, consisting of evenly distributed small spherical granules. The membranes of adjacent cells form a sharply defined net of perfectly polygonal mesh. The cytoplasm is stained a bright warm red or a paler pink by hæmatoxylin and eosin; in pyronin-methylgreen it is stained pale blue-green, but the membranes are stained pink. More deeply stained red particles sometimes lie on the pink membrane and project into the cytoplasm; rarely a similar particle lies isolated within the cytoplasm. I have not yet detected glycogen in the cytoplasm of these cells in normal material from the post-mortem room, but I have found it in similar cells in enlarged glands removed by operation. The nuclei measure from 3.5 to 7  $\mu$ , but 7  $\mu$  is very exceptional and 4 to 5  $\mu$  is usual. The smallest nuclei are found in the larger cells and are pyknotic. The pale oxyphil cells sometimes surround small alveolar spaces filled with acidophil coagulum.

The *principal cells* differ according to their degree of vesiculation. They vary in size from 6 to 15.5  $\mu$ . The smaller cells are least vesiculated. Their cytoplasm consists of an irregular net of particles of cytoplasm of different sizes. As vesiculation increases a cell-membrane becomes visible and the cytoplasmic net becomes ever more scanty until completely dropsical or ballooned cells are formed in which the nucleus lies free in a cavity bounded by a membrane (Fig. 183). The meshes of the net of membranes between adjacent ballooned cells are less perfectly polygonal, being more rounded, than those between pale oxyphil cells. The cytoplasm is stained a blue purple by hæmatoxylin and eosin. In pyronin-methylgreen the membrane and cytoplasmic net are pink, some large particles in the net usually being a deeper red. A very pale blue-green ground can be recognized in some cells. The principal cells are, therefore, essentially basophil. Glycogen is found in them, and is most abundant in the dropsical examples. The nuclei measure from 4 to 5.5  $\mu$ . They are usually lightly stained, but tend to be dark in the smaller cells. Alveolar spaces filled with acidophil coagulum are bounded more frequently by principal cells alone, or by principal cells and one or two dark or pale oxyphil cells, than by pale oxyphil cells alone. Those bounded by oxyphil cells alone are usually smaller.

*Nature of above Types of Cell.*—Although the dark and pale oxyphil cells

and the principal cells are easily differentiated, particularly in sections stained with pyronin-methylgreen, the three types appear to represent conditions of special activity, or conditions of exaggeration of a particular activity common to all, rather than to be cells of distinct origin and nature. Evidence of the origin of oxyphil cells from principal cells is found in the absence of oxyphil cells in the foetus and young child, the variation of their number in older subjects, and the sporadic presence among principal cells of single dark or pale oxyphil cells of similar shape and only slightly larger size. Further, pale oxyphil cells can apparently lose their peculiarities and become indistinguishable from ballooned glycogen-containing chief cells. Thus, not only are pale oxyphil cells often found within groups of ballooned cells, but sometimes in a nodule of pale oxyphil cells morphological transitions are found to completely dropsical cells with a basophil membrane from cells with a typical finely and evenly granular pale oxyphil cytoplasm and basophil membrane through cells with a similar cytoplasm broken to different degrees by large clear vesicles. There can be little doubt that dark and pale oxyphil cells are closely related. When cells of both types are present most of them lie in direct contact. Further, the depth of staining by eosin varies in different groups of pale oxyphil cells, the more deeply stained being of smaller size and having their cytoplasmic granules more closely packed. That oxyphil cells merely represent an exaggeration of an activity possessed by principal cells is suggested by the pale variety, and occasional examples of the dark, showing a basophil membrane, whilst a pale oxyphil substance can be detected in the basophil net of some principal cells. Further, the basophil cytoplasmic net of the principal cells usually contains more deeply basophil particles, whilst similar particles sometimes lie upon the basophil membrane of pale oxyphil cells and occasionally lie isolated within the oxyphil cytoplasm. The appearances suggest that the oxyphil cells are principal cells in which the cytoplasm has been so charged with oxyphil granules that the basophil net has been more or less completely reduced to a limiting membrane.

*The parathyroid glands in present case.*

In the present case the structure of the right parathyroid gland suggests an enlargement by an addition of lobules in the periphery rather than by a simple expansion. There is no evidence, however, that the enlargement has been associated with destruction of tissue or permeation of channels lined with endothelium. The structure can be explained by successive enlargement of the lobes of a segmented gland, a process which is taking place in the smaller parathyroid gland of Case 4. In both glands retrogression is indicated by hyaline degeneration of vessels. This is very slight in the left gland, but is considerable in the centre of the very large right gland, and is here complicated by calcification. Such retrogression is not proof of true neoplastic growth; severe degenerations occur in colloidal goitre, which is not definitely a neoplasm. The cells are of kinds found in normal glands: the only abnormalities are the cylindrical principal cells of unusual height and the giant nuclei. Cylindrical principal cells are found, however, in normal glands, and the mean diameter of these exceptionally elongated examples falls within normal limits. Giant nuclei are found in hyperplasias

that indicate abnormal functional activity; they are conspicuous in many thyroid glands in Graves' disease. There is, therefore, in the gross and minute structure of the two enlarged parathyroid glands nothing that indicates autonomous neoplasm rather than functional hyperactivity. In the hyperplasia there has been a very great increase of dark oxyphil cells; in the right gland their mass greatly exceeds that of all four glands in a normal subject. Principal cells in the dropsical or ballooned state form, however, a far greater proportion of the total mass of the two glands. Unfortunately no material was available for the detection of glycogen, which is usually abundant in ballooned principal cells.—H. M. T.

*Case 2.*—Hyperparathyroidism. Generalized osteitis fibrosa with multiple osteoclastomata. (Hunter, 1931).

M. F. R., single woman, aged 37. (L.H. Reg. No. 40737/1930.)

*HISTORY.*—Born in Greenwich. Employed in post office from 1908 (when aged 15) till March, 1928.

Summer, 1925.—While on holiday (chiefly walking) along Rhine valley, had slight injury to right foot making the arch painful for a few weeks. Continued to walk and was otherwise well.

May, 1926.—Slight pain in knees, hips, heels, and in arms. Weakness and lack of energy. Unable to walk any distance; had to give up tennis but was able to work. Occasionally cried with pain on shaking hands.

May, 1927.—Seven weeks' holiday on account of pain in legs and knees.

September.—Pain much worse in hips, knees, and heels. Legs and thighs unaffected. Pain never prevented her from sleeping, was worse when she moved about. In bed for seven weeks. Temperature was normal, but she was thought to be suffering from rheumatism. Pain and weakness occurred in the arms, and she had to give up work because she was unable to stoop or lift the heavy ledgers.

February, 1928.—Hardly able to get about because of pain and weakness. Unable to get up from a chair or to walk with aid of a stick. Slight swelling of jaw was noticed. Right hip was now the site of worst pain. Improved slightly, went back to work half-time for two months.

March.—Had to leave work permanently.

June.—Slipped from her chair and was doubled up at the hips. Had to be lifted up and has not walked since. Great stiffness of lower limbs, hips becoming very painful and knees remaining straight. Admitted to Greenwich and Deptford Hospital, where she remained in bed for five weeks. While under treatment she noticed a swelling of the left forearm and another on the back of the right hand. Mercury ointment was rubbed into these without effect. Radiograms taken of jaw and of both hip-joints. She was told that there was no fracture, but that the bones were very thin. Had massage at home for three months.

Oct. 28.—Admitted to Guy's Hospital. Weight, 5 st. 5 lb. 8 oz.

Nov. 14.—Accidentally thrown from a wheel-chair. Painful injury of right forearm.

*Radiograms of Bones.*—"All bones very translucent. Fracture of right radius and ulna. Shaft of left ulna shows cavitation and expansion of cortex. Ribs collapsed. Destruction of bone in third right metacarpal, both sides of mandible, necks of both femora and left iliac bone."

*Urine.*—Neutral; sp. gr., 1010; trace of albumin; Benee Jones protein absent; no sugar; deposit of phosphates.

*Blood-count.*—Red cells, 4,460,000; white cells, 15,000; polymorphonuclears, 70 per cent.

Feb. 1, 1929.—Portion of tumour of mandible removed for histological section. Easily cut with a knife. Dr. G. W. Nicholson reported upon the histology: "A giant-celled epulis".

Feb. 4.—Serum calcium 15 mgrm. per 100 c.c.; plasma phosphorus 3.3 mgrm. per 100 c.c.

Treatment: Right forearm immobilized in plaster for six weeks. Fractures united. Cod-liver oil given over a period of ten weeks in increasing doses, up to six drachms three times a day. During the last seven weeks of this treatment irradiated ergosterol was given; continued for three months, dose increased to 10 min. three times a day (radiostoleum). When the cod-liver oil was discontinued ultra-violet irradiation was used three times a week. Deep X-ray therapy applied to the bones (seven exposures).

March 5.—Left hospital.

April.—Noticed swelling on inner side of right leg below knee.

May 25.—Re-admitted to Guy's Hospital. Weight 4 st. 5 lb. 10 oz. Tumours of jaw and right hand larger.

*Radiograms of Bones.*—"Heads and necks of both femora appear to be absorbed and trochanters displaced upwards. Pelvis shows collapse and absorption of bone salts, more marked on right side."

Treatment: Four applications of deep X-ray therapy to jaw and arms. Insertion of radium into mouth.

July 5.—Left hospital.

October.—Had to give up writing and sewing.

April, 1930.—Re-admitted to Greenwich and Deptford Hospital. Further radiograms taken. Density of bone-shadows extremely poor. Many tumours expanding bone. Dr. Macdonald Critchley saw the patient in consultation with Dr. W. Denison Wiggins and diagnosed hyperparathyroidism.

*Past History.*—No history of rickets as a child. Catamenia began at age of 14; usually lasted a week. Normal until June, 1927, then somewhat excessive until June, 1928, when they suddenly ceased for eighteen months. Then irregular for six months and ceased entirely after April, 1930. In 1916 many teeth extracted for caries, leaving only eleven; in 1926, eight more teeth extracted. No history of polydipsia, polyuria, renal pain, or passage of stone or gravel. In 1923, while playing tennis, fell on outstretched hand and fractured left radius. Arm was in splint for ten days and bone united within a month.

*Family History.*—Father, one brother, and one sister, alive and well. Mother died from carcinoma of colon. No family history of fractures, dislocations, blue sclerotics, deafness, or peculiar shape of head.

May 28, 1930.—Admitted to the London Hospital.

ON EXAMINATION.—Height, 4 ft. 10 in. Weight, 4 st. 13 lb. Patient is intelligent and gives a concise history. Lies in bed totally disabled, deformed, frail, and wasted. Hips and knees flexed. Able to move left leg, but cannot straighten right leg. Unable to move either arm. Left arm aches. Great pain in right hip if she is lifted on that side. Pressure upon the bones, especially the forearms, gives rise to pain and this is severe when she is turned in bed. Sclerotics white. Lenses normal on examination by slit lamp. Hearing normal. Tongue clean. Mucous membranes normal colour. Thyroid gland normal. No tumour felt in neck. Forehead prominent, but skull otherwise normal. Hard, non-tender, bony enlargement on right side of mandible forming a swelling, 6 cm. in diameter. In the skin over it is a surgical scar. Tumour protrudes into mouth, where it is covered by bluish-red buccal mucosa. Only three teeth remain and appear to be normally calcified. Thorax tapers in upper part, superior inlet being thus reduced in diameter. Normal prominence of shoulders is missing, so that arms seem to disappear into upper part of chest, and clavicles to be too prominent. Lower thorax widens out, subcostal angle being normal. Eight slightly tender bony nodules felt in course of ribs, especially lower ribs. Slight scoliosis. Arms extremely thin, owing to muscular wasting and to diminution in total width of bones, are crossed upon chest; range of voluntary movement limited to a few inches. Antero-external curvature of both forearms. Upper end of left ulna presents a bony swelling. Large, slightly tender, bony swelling 4 cm. in diameter on dorsum of right hand apparently arising in third metacarpal. Second left metacarpal distorted and thickened. On passive

movement shoulder-joints are free, except in extreme abduction, which causes pain. Free flexion of elbow-joints is possible, but not full extension. Voluntary movements of left hip and knee are good. Marked abduction of left hip and complete extension of both knees give rise to pain. Movements of right hip limited. Sharp antero-posterior curvature of the upper end of right tibia and fibula with its concavity forward. Left lower limb not deformed. Bones of lower limbs not shortened.

CONTROL

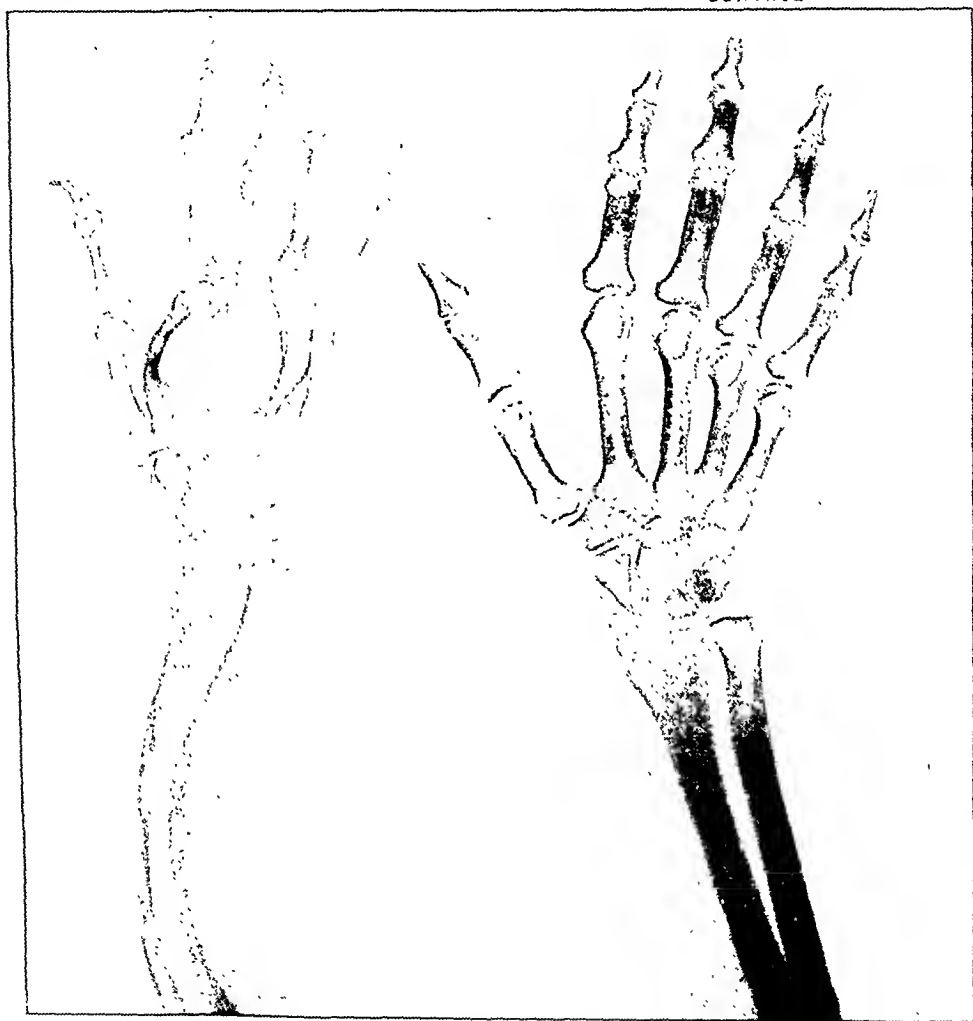


FIG. 178.—Case 2. Controlled radiogram of right forearm.  
(Reproduced by permission of the Royal Society of Medicine.)

Heart, lungs, and abdomen normal. Blood-pressure 140/100 mm. mercury. Generalized wasting and slight hypotonicity of muscles. No abnormality in nervous system. Trousseau and Chvostek signs absent. Wassermann reaction negative.

*Urine.*—Alkaline; sp. gr., 1014; albumin, heavy cloud; Bence Jones protein absent; no sugar. Deposit of amorphous and triple phosphates. Average daily output of urine (May–June, 1930) was 765 c.c.

*Renal Efficiency Tests.*—Blood urea 0.033 per cent.

		PHENOLSULFONEPHTHALEIN Percentage	UREA CONCENTRATION Percentage
First hour	.. 46	28.6	1.26
Second hour	.. 85	16.6	0.96



*Blood-count* (May 20, 1930).—Red cells, 4,400,000; Hb, 50 per cent; C.I., 0.55; leucocytes, 4920; differential count, normal; bleeding time and coagulation time, normal.

*Radiograms of Bones* (Fig. 178).—Greatly diminished density of shadows of all bones examined. This is emphasized by contrast with the bones of a control subject. Scoliosis. Collapse of chest wall with thinning of ribs and clavicles. Extremely diminished density of shadows throughout lumbar spine and pelvis. Both hips abnormal in position. The heads and necks of both femora have disappeared. Calvaria is mottled and shows two rounded pale areas with ill-defined

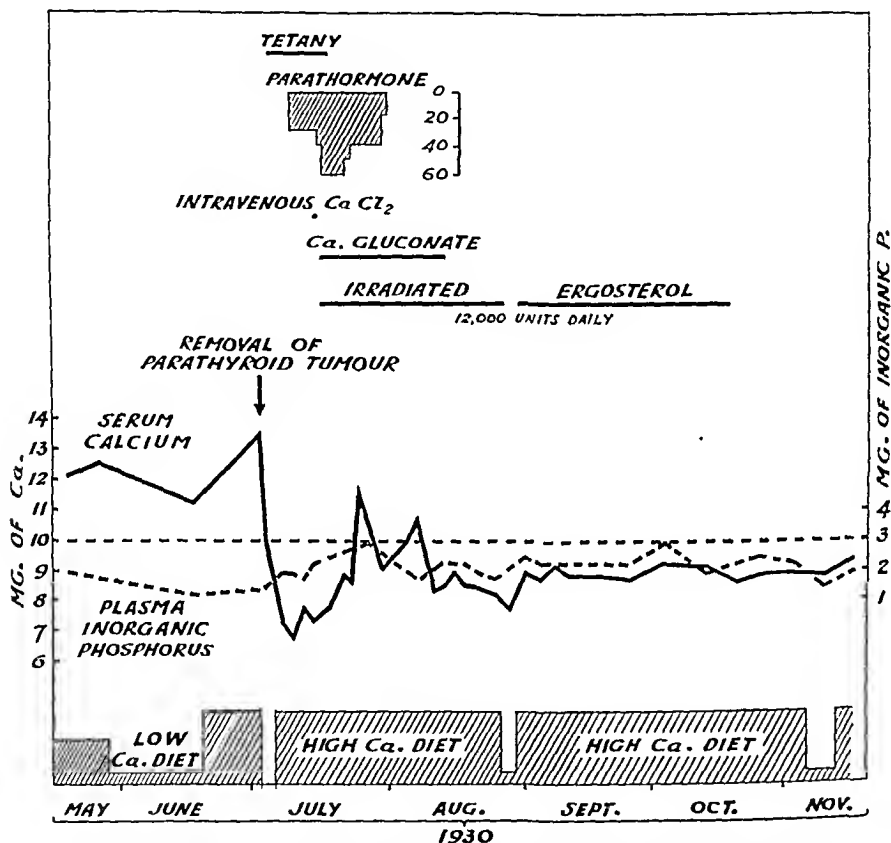


FIG. 179.—Case 2. Chemistry of the blood. The blood was examined at intervals for two months before and for five months after operation. The low calcium diet was that given during the investigation of the calcium balance. The high calcium diet was not weighed. It included four pints of milk daily, together with ice cream, cheese, and eggs. The scale indicating the dosage of parathormone represents units per day. The dosage of intravenous calcium chloride was 10 c.c. of a 5 per cent solution. The dosage of calcium gluconate was 10 c.c. of a 10 per cent solution daily, given by intramuscular injection. The dosage of irradiated ergosterol was measured in antirachitic rat units.

borders. Tumour of mandible dense and coarsely trabeculated. Head of right humerus has almost disappeared. Left humerus, right tibia, and right and left radius and ulna are bowed, frail and narrow, and show a grossly deficient corticallis. Irregular, pale, trabeculated areas expanding the corticallis are seen in right and left ulna, in several phalanges, and in second left and third right metacarpals. The last-named is so large as to distort the adjacent second and fourth metacarpals. Fracture of second metacarpal through a pale, cyst-like area at its mid-point.

**Radiograms of Kidneys.**—Kidney shadows normal in size. Two groups of small calculi seen in region of right kidney.

**Calcium and Phosphorus Metabolism before Operation.**—Hypercalcaemia was constantly present, the serum calcium varying between 11.2 and 13.6 mgrm. per 100 c.c. (Fig. 179). The corresponding figure for plasma phosphorus was 1.3 mgrm. per 100 c.c. Plasma phosphatase 1.13 mgrm. The calcium output was estimated in the urine and faeces for three three-day periods. The patient was kept on a weighed diet of known low calcium content. The calcium output in the urine was about three times the normal, that in the faeces being approximately normal (Fig. 180).

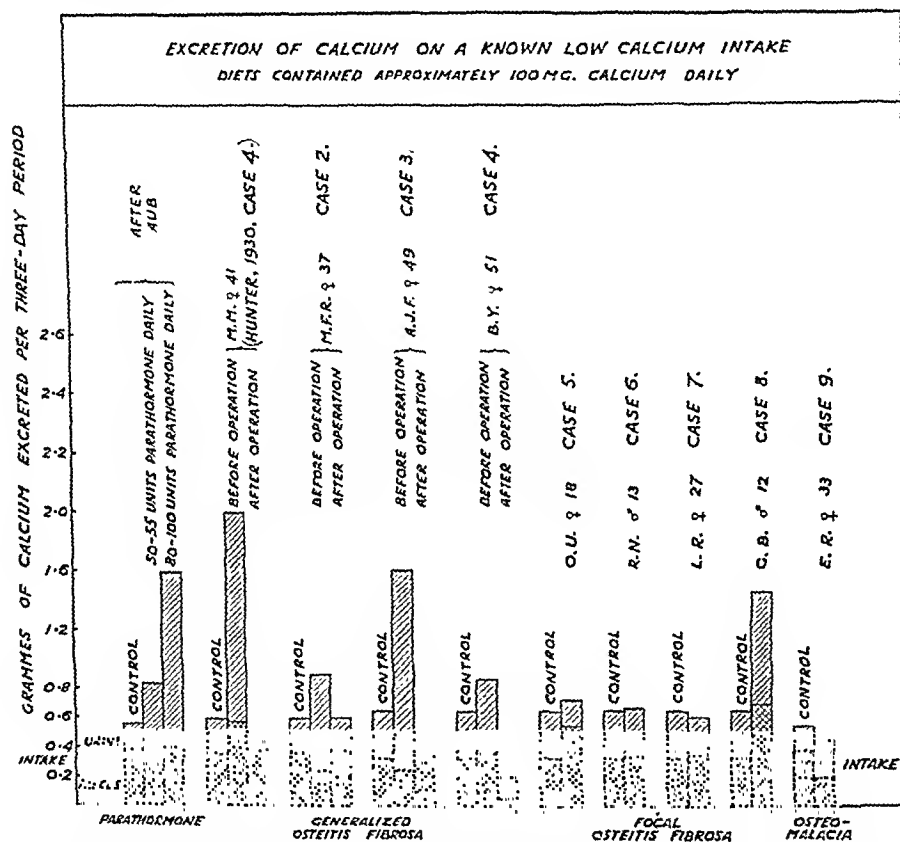


FIG. 180.—Calcium balance in generalized osteitis fibrosa, focal osteitis fibrosa, and osteomalacia. (The effects of injections of parathormone in normal individuals are shown in the first three columns.) The calcium excretion was estimated on a known low calcium intake. The horizontal lines represent the calcium intake, which was approximately 100 mgrm. daily. The urinary calcium excretion is represented by single cross-hatching and the faecal calcium excretion by double cross-hatching.

**OPERATION (Mr. A. J. Walton—July 2, 1930).**—Under open ether anaesthesia collar incision made in neck. Sternomastoid muscles were retracted on either side and pretracheal muscles divided. Thyroid gland apparently normal; inferior pole of left lobe was explored, and a normal left inferior parathyroid gland identified. The left superior parathyroid body, embedded in the posterior aspect of the upper pole of the left lobe of the thyroid gland, was dissected out with a blunt dissector. It was enlarged and measured approximately  $1.4 \times 0.8 \times 0.5$  cm. Its blood-supply was left intact. Exploration of the inferior pole of the right lobe of the thyroid gland failed to reveal the right inferior parathyroid body. An irregular projection from the lateral surface of the right lobe of the thyroid gland in its

upper part was dissected out and removed. As this was completed a tumour was unexpectedly displayed projecting from behind the trachea in the situation of the right superior parathyroid body. This was a smooth, solid, oval, brown-yellow tumour measuring  $2.3 \times 1.5 \times 0.9$  cm. It was removed. One drainage tube was inserted and the wound closed.

A portion of bone was removed from the upper part of the shaft of the right tibia.

### Pathological Report.

(PROFESSOR H. M. TURNBULL.)

Tissues removed at operation. (S.D. 1518/1930.)

#### 1. Right Tibia.—

**MACROSCOPIC.**—The specimen was a longitudinal slice from the upper part of the shaft. It was 3.5 cm. long. The outer border was covered with periosteum. In the lower 2 cm. the cut surface was 0.6 cm. wide and consisted of a gritty grey tissue, in which minute spicules of bone were seen with a hand-lens. It gave the impression of a closely-meshed spongiosa formed by very delicate trabeculae. In the upper 1.5 cm. the periosteal border bulged and was easily indented with a finger-nail. The cut surface was 1.1 cm. wide, and consisted of a rubbery grey gelatinous tissue in which no bone was seen. The slice consisted, therefore, of a lower, bony, and an upper, fleshy, segment. It was divided for microscopic examination through the bony part with great ease with a scalpel.

**MICROSCOPIC (Fig. 181).**—The two pieces were completely decalcified almost throughout with Müller's solution. In the lower, bony, segment there is no corticalis. The bone throughout consists of discrete branching trabeculae of bone in a relatively abundant fibrous marrow. The fibrous marrow is very uniform, and consists of closely-packed collagen fibrils, numerous spindle fibroblasts with oval nuclei, and vessels of normal structure. In places the vessels are dilated. Towards the medullary cavity the fibrous marrow encloses a very few small islands of adipose tissue. Any bony trabeculae that impinge upon this adipose tissue are surrounded by a zone of fibrous marrow. The trabeculae are slender throughout the bone, but they are specially slender in the subperiosteal third. In a few trabeculae towards the medulla there is a small portion of perfect lamellar bone. Otherwise the trabeculae consist of coarse-fibred and intermediate bone, and seldom contain more than one system. Active apposition is shown by numerous osteoid zones, on which the osteoblasts are large and close together. Where the osteoid zones are shown by the shape of the osteoblasts to have been cut strictly at right angles to their surface, they are not deeper than those seen in healing fractures. There are very few osteoclasts in Howship's lacunae until the upper, fleshy, part of the specimen is approached. Then many are seen in places, particularly in a narrow area immediately beneath the periosteum. In this area spindle fibroblasts are much more numerous in the marrow, groups of lymphocytes infiltrate the marrow, bony trabeculae are very few and small, and numerous osteoclasts lie upon or near them. Bone is being laid down, however, upon these trabeculae wherever osteoclasts are absent.

The upper, fleshy, segment is divided by strands of fibrotic marrow into lobular areas of cellular marrow crowded with giant cells. In the cellular areas spindle fibroblasts predominate, but there are many rounded, polygonal, and stellate forms. One karyokinetic figure was seen. Collagen and reticulum fibrils are usually scanty. There is a sparse infiltration with lymphocytes, monocytes, and occasional neutrophil leucocytes. Here and there are small groups of cells laden with iron pigment, and a diffuse impregnation with iron of the periphery of some lobules is shown by the Prussian-blue method. The vessels are capillaries bounded by a single or double row of cells. There are a few groups of extravasated red corpuscles, and more numerous very small areas of the fibrinous imbibition that, in association with extravasations, is so conspicuous a feature in most 'red tumours'. The giant cells resemble osteoclasts. They are very numerous, and most are very large. A few

are so deeply stained throughout that few of their nuclei can be recognized. Some have one or more large vacuoles in their cytoplasm, several contain an ingested monoocyte or neutrophil leucocyte, a few contain a rounded group of red corpuscles. The cellular marrow in the lobules merges into the fibrotic strands which separate the lobules from one another and from the periosteum. In these strands the marrow usually resembles that which predominates in the bony segment, but is more cellular

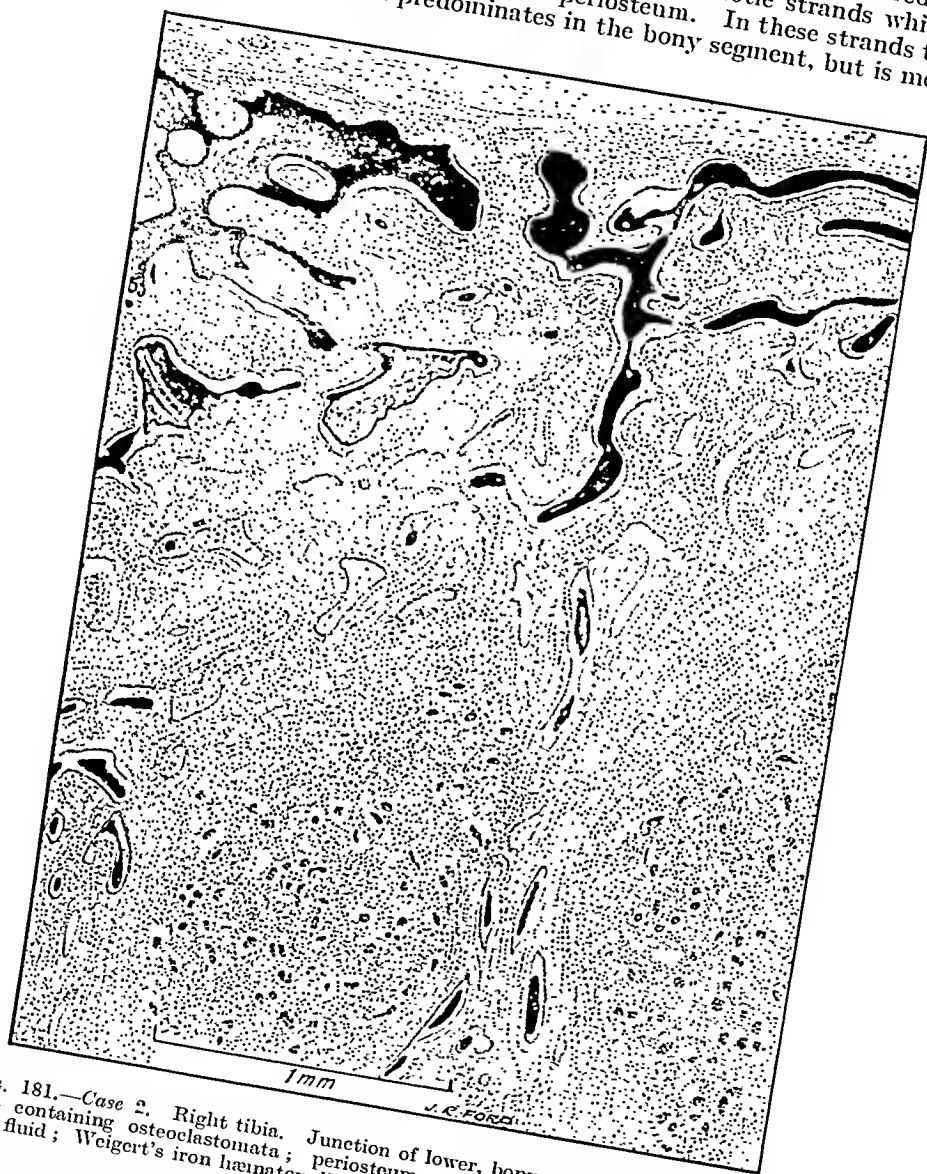


FIG. 181.—Case 2. Right tibia. Junction of lower, bony, segment with upper, fleshy, segment containing osteoclastomata; periosteum on top. Generalized osteitis fibrosa. Müller's fluid; Weigert's iron haematoxylin and van Gieson.

in the greater part of the strand beneath the periosteum. It usually contains trabeculae of woven bone bounded by osteoid tissue. Trabeculae of osteoid tissue alone are also seen, but comparison of serial sections shows these to be tangentially-cut osteoid zones upon calcified cores. The trabeculae resemble the more slender of those in the bony segment. The osteoid zones appear to be of similar depth. Numerous large osteoblasts lie upon the osteoid zones, but in the more cellular part

of the strand beneath the periosteum the zones of osteoblasts are frequently interrupted by osteoclasts in Howship's lacunæ. The lacunæ almost invariably indent osteoid zones and not calcified bone. The junction of the fleshy segment with the bony is sinuous, with concavities towards the bone.

**Remarks.**—The changes in the bone are essentially similar to those in *Case 1*. Great resorption is associated with apposition. All the normal bone appears to have been resorbed except a few microscopic pieces near the medulla of the lower, bony, segment. Trabeculæ of woven coarse-fibred and intermediate bone have been formed. These are almost all very slender and seldom show more than one system. Active apposition is in great excess of active resorption. Except in the neighbourhood of areas of more active cellular proliferation in the marrow active resorption is seldom seen and is slight. A conspicuous feature is the occurrence of active apposition by osteoblasts upon trabeculæ which are undergoing active resorption by osteoclasts in Howship's lacunæ. The depth of the osteoid zones is within the limits of the zones in healing fractures in healthy adults. There is again, therefore, no evidence of osteomalacia. The fibrosis of the marrow is again closely associated with apposition and resorption. Near the medulla in the lower segment the fibrosis is incomplete, small areas of marrow being still adipose. The trabeculæ that abut upon these areas are, however, bordered by a zone of fibrosis. In general it can be said that the fibrous marrow is more cellular where conspicuous resorption accompanies apposition than where apposition is alone seen, and that there is no bone in the areas of most cellular, least differentiated, fibrous marrow.

Bone in the upper segment is confined to septa of fibrous marrow which separate rounded areas of a very cellular marrow rich in osteoclasts. The osteoclasts are not now eroding bone, and they are so numerous, large, and evenly distributed that it does not appear justifiable to attribute their presence to recent resorptive activity. These cellular areas have caused expansion of the bone. Histologically they cannot be differentiated from the less anaplastic examples in a series of osteogenetic myelomata, or osteoclastomata, removed from the ends of long bones in skeletons free from general changes. In a nomenclature depending upon morphology, therefore, such areas are entitled to the name 'osteoclastoma', although their retrogression following excision of enlarged parathyroid glands points to their not being autonomous neoplasms.—H. M. T.



FIG. 182.—*Case 2*. Generalized osteitis fibrosa. Tumour of right superior parathyroid body.  
(Actual size.)

## 2. Right Superior Parathyroid Body.—

**MACROSCOPIC.**—An oval body, measuring  $2.3 \times 1.5 \times 0.9$  cm. and weighing 1.3 gm. (*Fig. 182*). One surface was convex, the other slightly concave. Externally it was brown-yellow, even, and smooth except where a cord of areolar tissue was attached near one pole. The cut surface was homogeneous, moist, and paler brown-yellow. The cord of areolar tissue attached it to a mass ( $2 \times 1.2 \times 0.6$  cm.) which was darker brown and had a slightly nodular surface. The cut surface was a considerably darker brown than that of the oval mass, and glistened conspicuously.

**MICROSCOPIC.**—The oval body is a parathyroid gland. It has a thin capsule of collagenous and elastic fibres, and is divided by a net of delicate collagenous

fibrils (Laidlaw's and van Gieson's stains) into small acini (*Fig. 183*). The inter-acinar septa contain capillaries, venous capillaries, or veins. Most of the acini are filled with mutually compressed cells, but a few have a central sphere of oxyphil coagulum surrounded by a row of cubical cells. Giant nuclei and multiple nuclei are conspicuous features. Most of the cells are completely dropsical or ballooned principal cells in which the cytoplasm is reduced to a basophil membrane. These measure from 7 to 23  $\mu$  in mean diameter. Most, however, are from 11 to 12  $\mu$  with a nucleus of 6  $\mu$ . In the larger forms the nuclei are from 6 to 19  $\mu$ , and some larger forms have two nuclei. Almost all the nuclei are deeply stained, but show a close net of chromatin, numerous nodes, and one to six nucleoli. In places are a few principal cells with a reticular basophil cytoplasm. They measure 7 to 13  $\mu$ , usually

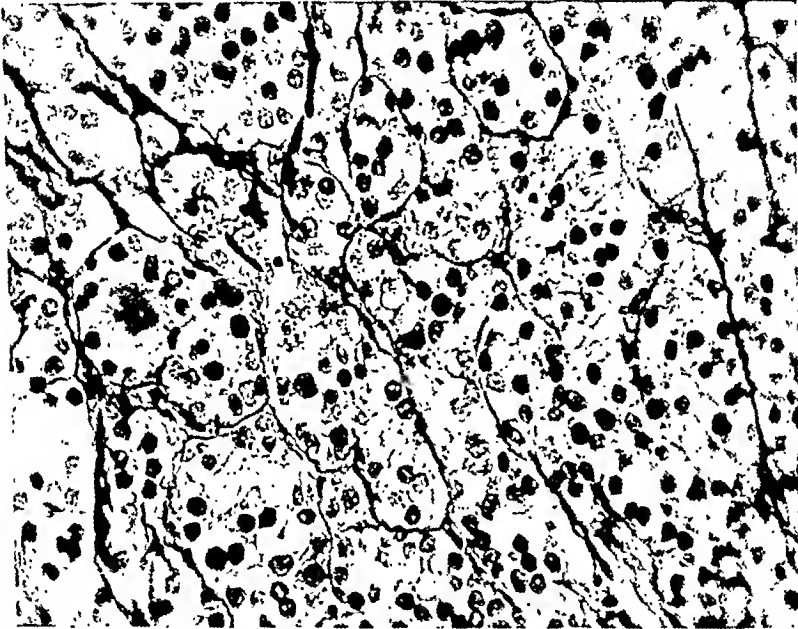


FIG. 183.—Case 2. Enlarged parathyroid gland. Generalized osteitis fibrosa. Acini containing ballooned principal cells; in one there is a lumen filled with coagulum. Laidlaw's collagen-reticulum silver stain. ( $\times 320$ .)

9 to 10  $\mu$ . The nuclei average 6  $\mu$  and are either lightly or deeply stained. In those in which vesiculation is marked a pale oxyphil substance usually lies in the meshes of the remnant of basophil reticulum within a basophil membrane. One or two oxyphil cells are seen in each alveolus in one small area, but elsewhere they are very scanty. They are almost all of the pale oxyphil type with a basophil membrane. Most measure from 12 to 13  $\mu$ , with nuclei of 6 to 7  $\mu$ ; but there are a very few small examples of 9.5  $\mu$  and several large forms up to 25  $\mu$ . The largest nucleus measures 8  $\mu$ , but several of the larger cells have two nuclei and one has four. Almost all the cells that surround coagulum are principal cells. Herxheimer's method shows one or two lightly stained spheres, up to 3  $\mu$  in diameter, within ballooned cells. Best's method shows abundant glycogen within the ballooned cells. It is sometimes present in scattered droplets, but more often fills the cell or forms a crescent in the periphery.

The body attached to the parathyroid gland consists chiefly of a rounded nodule of slightly fibrotic thyroid tissue. Almost all the acini contain a thin colloid and are bounded by a row of well-defined flattened cubical cells. A few acini are greatly distended with this colloid. A very few small acini are lined with larger ill-defined cells, and appear to be in an early stage of colloid secretion. There are a

few groups of lymphocytes, with occasional plasma cells, in lymphatic sinuses. This nodule is separated from the septum from a strip of thyroid tissue. The strip differs in that fibrosis is confined to the neighbourhood of the septum, distension with colloid is not so great, and there is only one minute area of lymphocytic infiltration.

**Remarks.**—The only abnormality in the parathyroid body besides enlargement is the presence of giant nuclei and multiple nuclei. The giant nuclei are numerous, and are very conspicuous under a low power. Giant and multiple nuclei are sometimes as conspicuous in the thyroid gland of Graves' disease. The appearances suggest an exceptional functional activity. The hyperplasia resembles that in *Case 1* in that it consists chiefly of ballooned principal cells; these cells contain much glycogen. Oxyphil cells are, however, relatively much more scanty than in *Case 1*, and they are almost all of the pale variety. Further, the acini are smaller and more completely limited (*Fig. 183*).

If the portion of thyroid gland examined can be taken to represent the whole, the gland shows a greater activity than is normal at the age of the patient.—H. M. T.

**SUBSEQUENT PROGRESS.**—Twenty-two hours after operation Chvostek sign was positive. On the second day tinglings occurred in the fingers, and the bones were found to be less tender on pressure. On the third day the patient complained of cramps in the fingers, hands, feet, and trunk, so that she felt 'tied in knots'. On the fourth day, and subsequently, passive movements of the limbs and pressure on the bones caused no pain. Immediately after operation a diet containing milk, ice-cream, and eggs was given, together with calcium lactate (5 gm. three times a day). In spite of the high calcium intake, cramps in the limbs persisted, and parathormone was given by intramuscular injection from the sixth day for twenty-six days (*Fig. 179*). Intramuscular calcium gluconate (10 c.c. of a 10 per cent solution) was used to relieve tetany, and on one occasion intravenous calcium chloride (10 c.c. of a 5 per cent solution) was required (*Fig. 179*). Irradiated ergosterol (one radiostol tablet twice a day) was given over a long period. On the tenth day after operation acuity of hearing became remarkably increased and remained so for five days; subsequently hearing was normal. All manifestations of tetany ceased on the seventeenth day after operation. On the nineteenth day she began to be alert and interested, and found that she could turn over the pages of a book. Five weeks after operation the bony tumour on the dorsum of the right hand had almost disappeared, and the swelling inside the mouth, corresponding to the tumour on the mandible, was much smaller. In six weeks, after treatment by massage, she was able to move her arms down to her sides. In eight weeks she was lifted into a chair and soon became accustomed to sit comfortably. Three months after operation normal menstruation occurred. Muscular development improved. Five months after operation she stood and began to walk with assistance. In seven months she began to knit, to sew, and to write. In nine months she walked with crutches and was soon able to use them to climb up and down a few steps. Ten months after operation the weight had increased by 24 lb. (6 st. 9 lb. on April 27, 1931).

**Radiograms** (Nov. 24, 1930).—Density of bone shadows, as compared with controls, is not appreciably different from what was seen before operation. Cyst-like areas expanding bone are smaller, especially in the third right metacarpal, and some of them appear to be filling in. Renal calculi unaltered.

**Blood-count** (May 4, 1931).—Red cells, 4,000,000; Hb., 50 per cent; C.I., 0.62; leucocytes, 8000; differential count, normal.

**Urine** (Dec. 2, 1930).—Acid; sp. gr., 1018; albumin, faint trace; no sugar; deposit, amorphous urates. The average daily output of urine (July–November, 1930) was 570 c.c.

*Calcium and Phosphorus Metabolism after Operation.*—After parathormone injections were discontinued the serum calcium remained slightly below the normal level, namely 9.0 mgrm. per 100 c.c., and the plasma phosphorus rose to above 2.0 mgrm. per 100 c.c. (Fig. 179). Latent tetany disappeared. The plasma phosphatase remained high, the average of 13 readings after operation being 0.73 mgrm. Four months after operation the calcium output was estimated for two three-day periods on a low calcium diet. The urinary output of calcium had dropped to half the pre-operative level, but was still twice that of the normal control (Fig. 180).

**Remarks.**—Gold (1928) was the first to demonstrate hyperparathyroidism in a case of generalized osteitis fibrosa, associated with multiple tumours and cysts of bone. Similar cases, in which bone tumours were obvious on clinical examination, have since been recorded (Barr, Bulger, and Dixon, 1929; Wilder, 1929). Renal calculi have often been demonstrated in hyperparathyroidism, and the present case shows that persistent slight albuminuria may be the only indication of their presence. Generalized osteitis fibrosa has sometimes been treated with irradiated ergosterol in the hope of increasing the apposition of bone (Regnier, 1929). In at least one case (Snapper and Boevé, 1931) good evidence has been obtained of the effect of such treatment in increasing the density of bone as judged by radiograms. In the present case no improvement occurred even with the prolonged use of large doses of cod-liver oil, irradiated ergosterol, and ultra-violet irradiation.

The remarkable improvement after removal of a parathyroid tumour is of great interest. The patient has been changed from a pain-racked bed-ridden cripple to an almost normal individual. We must not forget, however, that the urinary calcium output, as estimated four months after operation, was still in excess of the normal, and it is possible that hyperparathyroidism still exists and may even cause a return of symptoms. Decrease in size of osteoclastomata after removal of a parathyroid tumour was first recorded by Barr, Bulger, and Dixon in 1929. Restoration to normal of the menstrual function has also been previously recorded. The temporary occurrence of hyperaemia following the operation is of interest. It has been observed where the serum calcium was low from another cause.

**Case 3.**—Hyperparathyroidism. Generalized osteitis fibrosa. Bilateral renal calculi.

A. J. F., married woman, aged 49. (L.H. Reg. No. 41234/1930.)

**HISTORY.**—1919: Onset of thirst, dry mouth, and polyuria. Began to take two quarts of water or lemonade during the night.

1922.—Attack of symptomless hæmaturia, blood and pus found in the urine, bilateral renal calculi demonstrated in radiograms. Operation (Mr. F. S. Batchelor, Dunedin): right nephrotomy—removal of large renal calculus. Temporary relief of thirst. Three years after this operation began to have severe attacks of renal colic, which have persisted until the present time.

1927.—Became unable to walk long distances owing to fatigue. Noticed a hard lump on the right shin, which grew larger and became tender. Right leg stiff and painful, unable to climb steps. Pain around joints, for which all teeth were extracted. Operation (Mr. F. S. Batchelor): removal of portion of bone tumour for histological section. Report: "portion of bone from right tibia shows matrix of fibroblasts and threads of fibrous tissue, also fragments of osteoid tissue. Many osteoclasts are present in the matrix and applied to many of the osteoid fragments. There is also blood pigment from old hæmorrhages. The appearances



are those seen in the cystic areas of osteitis fibrosa cystica (von Recklinghausen's disease).”—Dr. Murray Drennan (Dunedin).

Following this operation outward bowing of the right thigh occurred. The patient wore a splint for two years, removing it when she went to bed. There was pain in the left hip and groin after walking any distance. Both legs became very tender, and at one time she could not bear anything to touch them. Radiograms of the bones were taken every three months, and she was told that some of the swellings in the bones disappeared, and fresh ones appeared elsewhere. Treatment with preparations of vitamin D and with ultra-violet irradiation had some good effect.

November, 1929.—Discarded caliper splint. Less pain in bones, still unable to mount stairs. Walked with a stick, but could manage half a mile with ease.



FIG. 184.—Case 3. Radiogram of right kidney.

*Past History.*—Onset of catamenia at 14; lasted five or six days; regular every month until a year ago, then suddenly ceased. Five children alive and well. One miscarriage at three months in 1918; stillborn child in 1919, at onset of first symptoms of illness. In each case labour was normal. Babies all breast-fed until nine months. For many years past had had repeated dental treatment; six teeth were crowned. There was no dental caries; the teeth were removed on the assumption that they caused rheumatism. Has never fractured a bone. No family history suggesting disease of bones.

Aug. 22, 1930.—Admitted to the London Hospital.

ON EXAMINATION.—Height 5 ft. 4 in. Weight 9 stone. Well-nourished woman, gets about well, prefers to use a stick. Calvaria of normal size and contour. Sclerotics white. Pupils equal and regular, react to light and on accommodation. Right and left coronary cataract with some small scattered opacities in the anterior cortex (Mr. C. Goulden). Hearing normal. Double dentures. Tongue clean. Mucous membranes of normal colour. Small, firm adenoma 2·7 cm. diam. in lower pole of left lobe of thyroid gland. No other tumour felt in neck. Heart and lungs normal. Blood pressure 120/90 mm. mercury. No hypotonicity of muscles. No abnormality in nervous system. Trousseau and Chvostek signs absent. No tenderness on pressure upon shafts of bones. Slightly raised, hard, non-tender, bony swelling on inner aspect of right tibia 4 cm. diam., covered by scar of operation. Depression felt in subcutaneous surface of left tibia immediately below left knee-joint. No other bone deformities nor tumour. Nephrotomy scar in right loin.

*Urine.*—Acid; sp. gr., 1005; albumin, heavy cloud; no Benec Jones protein; deposit, leucocytes and red cells.

*Blood.*—Blood-count, bleeding time, and coagulation time normal. Wassermann reaction negative.



FIG. 185.—Case 3. Radiogram of skull.

*Radiograms.*—Bilateral renal calculi (Fig. 184). Pale, rounded cyst-like areas in lower end of right femur, both tibiae, metacarpals, phalanges of fingers, bones of pelvis, and calvaria (Fig. 185). Slight diminution of density of shadows of bones as compared with a normal control.

*Calcium and Phosphorus Metabolism before Operation.*—Hypercalcaemia was constantly present, the serum calcium reaching as high as 15·0 mgrm. per 100 c.c. The plasma phosphorus was low, namely, 1·8 mgrm. per 100 c.c. (Fig. 186). Plasma phosphatase, 1·16 mgrm. For three three-day periods on a known low calcium intake the calcium output in the urine was approximately four times the normal, that in the faeces being normal (Fig. 180).

OPERATION (Mr. A. J. Walton—Aug. 29, 1930).—Under open ether anaesthesia

a collar incision was made in the neck. Small adenoma (2.7 cm. diam.) in lower pole of left lobe of thyroid gland. Behind this was a tumour 6.8 cm. long lying between the trachea and the œsophagus with its lower pole in the mediastinum. It was easily dislocated upwards, and was found to have a vascular pedicle attached to the inferior thyroid artery. The pedicle was ligatured and the tumour

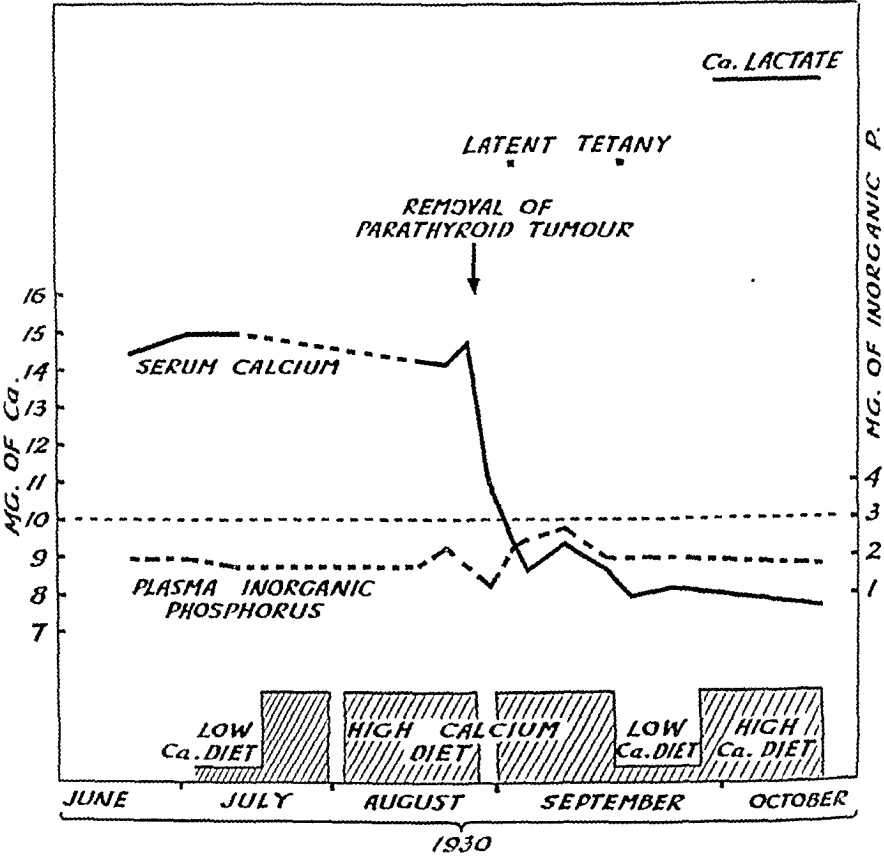


FIG. 186.—Case 3. Chemistry of the blood. The blood was examined at intervals for three months before and for two months after operation. The low calcium diet was that given during the investigation of the calcium balance. The high calcium diet was not weighed. It included four pints of milk daily, together with ice cream, cheese, and eggs.

removed. No other parathyroid bodies were disturbed. The adenoma was shelled out from the left lobe of the thyroid gland, which was then sutured. An incision was made over the bony tumour in the right tibia. A portion of tumour together with the corticalis below it was removed with the periosteum intact.

Pathological Report.

(PROFESSOR H. M. TURNBULL.)

Tissues removed at operation. (S.D. 2003/1930.)

1. Right Tibia.—

MACROSCOPIC.—The specimen was a vertical slice covered with periosteum and measuring 3.5 cm. long. The cut surface showed beneath the periosteum three successive kinds of tissue. In the upper 1.8 cm., which corresponded to the swelling seen upon the tibia, it was 0.7 cm. wide and was occupied by a soft tissue, which was



FIG. 187.—Case 3. Right tibia, central segment. Generalized osteitis fibrosa. Note the minute osteoclastoma, and compare the general changes with those in the example of focal osteitis fibrosa (Fig. 188). Müller's fluid followed by nitric acid; Weigert's iron-haematoxylin and van Gieson.

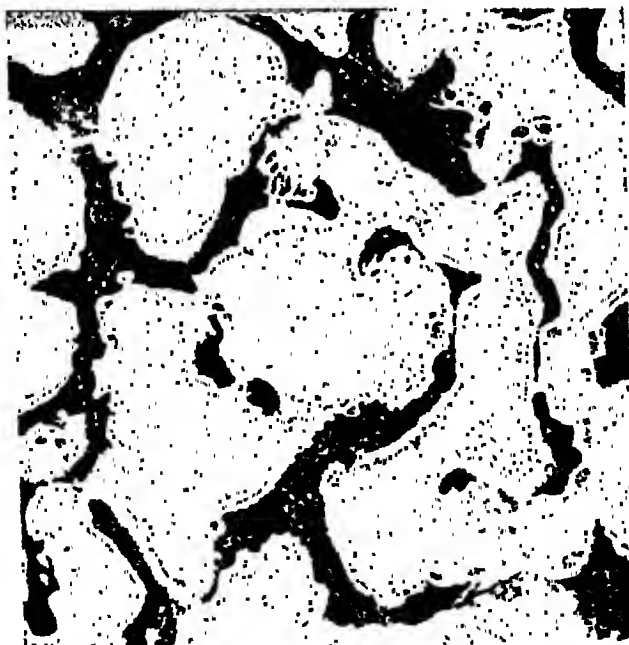


FIG. 188.—Case 8. Right femur. Osteitis fibrosa in multiple foci. Müller's fluid, followed by nitric acid; Weigert's iron-haematoxylin and van Gieson.

mottled grey-brown and mahogany-brown and was separated from the periosteum by a very thin interrupted sheet of gritty bone. The central 1.1 cm. was also 0.7 cm. wide, could be cut without difficulty with a knife, and showed a close mesh-work of delicate trabeculae of bone enclosing yellow-brown tissue. The lower 0.6 cm. was 0.5 cm. wide and consisted of a grey bone which differed from ivory bone only in being stippled with dark points. The edge of this dense bone where it met the spongy was concave and sharply defined. The junction of the spongy bone with the soft tissue was sinuous and less sharply defined.

**MICROSCOPIC.**—The bone was fully decalcified in Müller's solution followed by nitric acid. In the lower of the three segments described above the outer two-thirds of the corticalis are abnormal only in that there are many small medullary spaces throughout and a few bays in the periosteal surface, which communicate with medullary spaces. The bone consists of systems of lamellar bone which enclose areas and systems of woven bone. Active resorption is shown by numerous osteoclasts in Howship's lacunae in the margins of some of the medullary spaces. Active apposition preponderates, however, most of the medullary spaces being lined with an osteoid zone covered by closely-packed large osteoblasts. The medullary spaces contain a fibrous marrow. In the inner third of the corticalis the bone is reduced to a few narrow trabeculae, which lie in a fibrous marrow that surrounds a small rounded osteoclastoma. The trabeculae are undergoing active resorption and apposition. The latter predominates and has led to the formation of some small processes of woven bone.

In the central segment (*Fig. 187*) the medullary spaces in the outer two-thirds of the corticalis are larger and have anastomosed so that the compacta is converted into a finely meshed spongiosa. The medullary spaces are of very irregular shape. Their longer axis is most often at right angles to the long axis of the bone. In some fields the bone is in slight excess of the soft tissue, in others the soft tissue is in great excess. In one of the larger medullary spaces is a minute osteoclastoma (*Fig. 187*). The trabeculae consist chiefly of lamellar bone, but this encloses numerous areas and systems of woven bone. Similar woven bone has been formed in places immediately beneath a stout tendinous insertion upon the outer surface, and the inclusions beneath doubtless represent periosteal interstitial systems that have been incorporated in the growth of the original compacta. Active apposition is present almost throughout and is in great excess of resorption, but numerous osteoclasts in lacunae are present in a few places. The medullary spaces contain a fibrous marrow. In the inner third of the corticalis the bone is again reduced to a few scattered slender trabeculae in a zone of fibrous marrow which surrounds two osteoclastomata.

In the upper segment the corticalis is reduced to a few trabeculae of very irregular shape. These lie in a fibrous marrow that forms septa between, and to a less extent within, large rounded osteoclastomata and also separates the osteoclastomata in places from the periosteum. Most of the trabeculae consist of lamellar bone, but towards the osteoclastomata are a few very slender trabeculae of recently formed woven bone. Active resorption is shown by many osteoclasts in lacunae in osteoid tissue or in bone, but active apposition is again in great excess.

In all segments the osteoid zones are deep. They appear to increase in depth the more the corticalis has been eroded. This apparent increase in depth is due to tangential section being ever more frequent the more irregular the outline of the trabeculae. Where the zones are cut at right angles to their surface the depth is within the limits of that found in actively healing fractures. The fibrous marrow is similar to that described in other specimens. It varies slightly in density. The less dense areas contain very wide capillaries, and lie chiefly near the large osteoclastomata. Cells loaded with iron-pigment are numerous throughout; they form dense masses near the osteoclastomata. The rounded areas called osteoclastomata are very similar to those that have been described in *Case 2*. They differ in that there is very great extravasation of red corpuscles within them and immediately round them.

**Remarks.**—A great resorption of bone is associated with a fibrotic marrow that contains osteoclastomata—that is to say, areas of greater cellularity containing great numbers of large osteoclasts. In the osteoclastomata there is no bone, in the fibrotic marrow at the margins of the osteoclastomata the corticalis has been reduced to a few slender trabeculae. Farther from the osteoclastomata there is a porosity of the compacta, which is very great near the group of larger osteoclastomata but becomes less at a distance. Resorption of bone, therefore, has been very great and has caused the main change in structure. Lacunar resorption is still active in places, but apposition of bone is now the predominant activity. The apposition is taking place rapidly and the osteoid zones are correspondingly deep, but there is no evidence of a delay in calcification such as occurs in rickets and osteomalacia. This case differs from the first two in that most of the bone present, and almost all where the porosity is least, appears to be a remnant of the original, normal compacta. The abnormal processes seen in the first two cases are here in an earlier stage.—H. M. T.

## 2. Left Inferior Parathyroid Gland.—

**MACROSCOPIC (Fig. 189).**—The specimen was spindle-shaped, with one convex and one slightly concave surface, measured  $6.8 \times 2.8 \times 1.4$  cm., and weighed 13.5 gm. The outer surface showed a few, irregularly scattered shallow sulci, in one of which blood-vessels entered the gland. It was covered with a transparent capsule. In about one half of the cut surface a yellowish-brown tissue enclosed numerous small cysts (from 0.2 to 1 cm. in diameter), containing clear fluid. In the other half the yellow-brown tissue was ragged and spongy owing to numerous cysts with ill-defined, irregular boundaries.

**MICROSCOPIC.**—The gland is surrounded by a thin sheet of collagenous and elastic fibres, and is imperfectly divided into acini by numerous capillaries or larger vessels sheathed by collagenous fibrils. No other reticulum is shown by Laidlaw's silver impregnation. In three of the six portions taken for microscopic examination there are a few small acini of principal cells immediately under the capsule. The cells measure from 7 to 9  $\mu$  in mean diameter. They have a basophil reticular cytoplasm, no visible cell-membrane, and a nucleus of 4 to 5  $\mu$ , which is usually deeply stained. All the rest of the gland is occupied by oxyphil cells. These are almost all pale oxyphil cells (Fig. 190).

They measure from 9 to 19  $\mu$ , but most are 14 to 15  $\mu$ . They are polygonal from mutual compression, and their basophil cell-membranes form a conspicuous polygonal intercellular net. The oxyphil cytoplasmic granules are set closely in some cells, but in the majority they are unusually wide apart, many cells having a very dropsical appearance. The nuclei measure from 4.5 to 6  $\mu$ , are usually lightly stained, and occasionally show one or two inconspicuous nucleoli. Lysis, accompanied by shrinkage, of nuclei is frequent; one

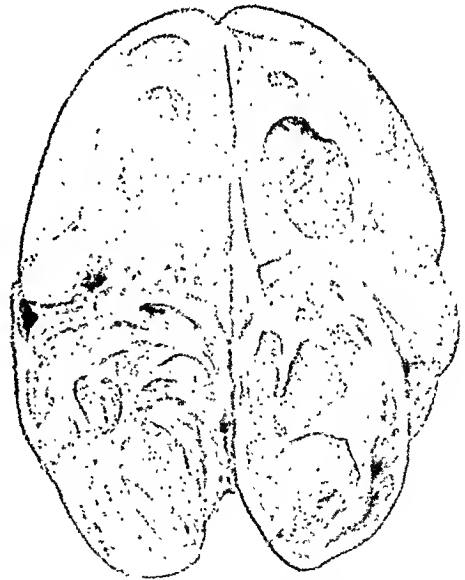


FIG. 189.—Case 3. Generalized osteitis fibrosa. Tumour of left inferior parathyroid body. (Actual size.)

karyokinetic figure was seen. Dark oxyphil cells are scanty, but lie here and there, compressed between pale oxyphil cells. They measure from  $6.5$  to  $8.5 \mu$ , and have a nucleus of  $4$  to  $4.5 \mu$ . The nuclei are deeply stained, and usually narrow-oval. In tissue fixed in alcohol and stained by Best's method almost all the pale oxyphil cells contain glycogen. The glycogen is usually in the form of small granules scattered throughout the cell, but many cells contain larger droplets and irregular masses, which usually lie near the periphery of the cell, often forming

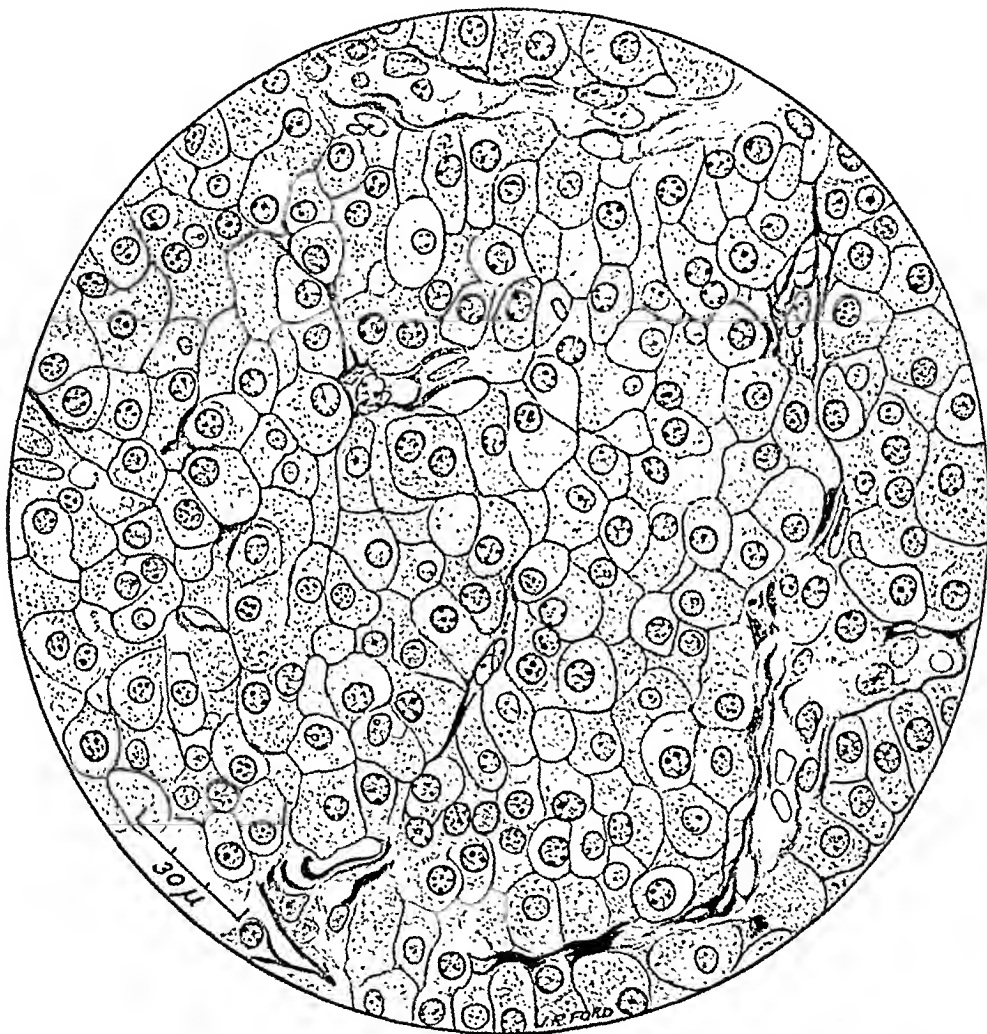


FIG. 190.—Case 3. Enlarged parathyroid gland. Generalized osteitis fibrosa. Pale oxyphil cells, with granules separated to various degrees by dropsy. Weigert's iron-haematoxylin and van Gieson.

a crescent within the cell membrane. Glycogen is, therefore, very abundant, but does not fill these cells so completely as it fills the ballooned principal cells of Case 2. No fat is shown by Herxheimer's method, but many cells contain very lightly stained refractile granules similar to, but usually much smaller than, those seen in previous cases. From their size and position they appear to be granules of glycogen that have survived fixation in formaldehyde. Even in the more solid parts of the gland there are a few clefts or larger cystic spaces within the acini.

In the spongy parts such cysts are very numerous and are often of large size. The number of cells in the walls of the cysts usually varies in different parts of the circumference. The inner margin of the smaller cysts is irregular, in the larger it is even. The cysts contain a thin oxyphil, usually bubbly coagulum, in which there are occasionally degenerated epithelial cells and rarely a few red corpuscles. Droplets of glycogen are occasionally present, chiefly within degenerated cells. The vascular septa are occasionally distended by a similar coagulum; more often they are distended by œdema. Within the septa are a few tissue-mast-cells; a few lymphocytes and one or two plasma cells were also found.

### 3. A Body Excised from Lower Pole of Left Lobe of Thyroid Gland.—

**MACROSCOPIC AND MICROSCOPIC.**—A spherical, encapsulated colloidal adenoma of the thyroid gland, showing cystic and atheromatous degeneration and extensive hyaline fibrosis. It measured 2.7 cm. in diameter, and weighed 8.8 gm.

**Remarks.**—In the enlarged parathyroid body the cells and their arrangement are in no way atypical. The appearances suggest a hyperplasia and excessive secretory activity rather than an autonomous neoplasm. Here and there beneath the capsule are a few small acini containing principal cells. All the rest of the gland is occupied by oxyphil cells. The enlargement contrasts, therefore, sharply with that of *Case 2*, which consisted almost entirely of principal cells, and less sharply with that of *Case 1*, in which oxyphil cells took a relatively small, though actually large, share. Here, however, unlike *Case 1*, the oxyphil cells are almost all of the pale variety, and in general they show much dropsical separation of the oxyphil granules (*Fig. 190*). They contain much glycogen, but are not so filled with glycogen as the ballooned principal cells of *Case 2*. Cystic spaces filled with thin oxyphil secretion are very numerous, and this secretion also infiltrates the septa in places.—H. M. T.

**SUBSEQUENT PROGRESS.**—On the first day after operation the patient complained of feelings of pins and needles in the hands. No manifest tetany occurred. Trousseau sign was negative and Chvostek sign was only twice elicited. On the fifth day after operation the serum calcium dropped to 8.7 mgrm. per 100 c.c. (*Fig. 186*). At this time she was feeling well, thirst had completely disappeared, and there was no longer any aching in the limbs. Radiograms of the kidneys showed no change in the shadows of the renal calculi. Ten months after operation the patient was perfectly well, had gained 2 st. 9 lb. in weight, and could walk four miles without fatigue. Thirst and aching in the bones had disappeared.

**Calcium and Phosphorus Metabolism after Operation.**—A diet high in calcium content was resumed after operation; 15 gm. of calcium lactate were given daily. Intramuscular calcium gluconate and parathormone injections were unnecessary. Three weeks after operation the calcium output was estimated for three three-day periods on the same low calcium diet as before. The output in the urine was very much less than in a normal individual; that in the faeces was normal (*Fig. 180*). Four weeks after operation the plasma phosphatase was 1.2 mgrm. Ten months after operation the serum calcium was 10.6 mgrm. per 100 c.c., and the plasma phosphorus 3.8 mgrm. per 100 c.c.

**Remarks.**—The spontaneous remission recorded in this case suggests that hyperparathyroidism may sometimes show relapses and remissions analogous to those seen in acromegaly and exophthalmic goitre.

Where four times the normal amount of calcium is excreted in the urine it is not surprising to find polyuria. Thirst and polyuria are mentioned in



five published cases of hyperparathyroidism. Both symptoms are usually promptly abolished after removal of the parathyroid tumour (Boyd, Milgram, and Stearns, 1929; Pemberton and Geddie, 1930). Some authors refer to the polydipsia and polyuria as diabetes insipidus (Rosenbach and Disqué, 1930).

Renal calculi have been recorded in ten cases, and renal colic was the presenting symptom in at least one case (Léri, Layani, Lièvre, and Weill, 1930). In one case renal calculi began to break up a few weeks after operation and were passed into the bladder (Hurst and Cosin, 1930).

*Case 4.*—Hyperparathyroidism. Generalized osteitis fibrosa with multiple osteoclastomata. Otosclerosis.

B. Y., married woman, aged 51. (L.H. Reg. No. 41770/1930.)

HISTORY.—1892: Acute febrile illness with severe pain in left hip followed by formation of an abscess which discharged profusely.

1893.—Painless swelling on upper part of right arm; discharge of pus and sequestrum.

1894.—Operation upon left hip for scraping of bone. Wound healed temporarily.

1895.—First noticed deafness. No pain. No otorrhœa.

1896.—Swelling of right hip resulting in formation of a sinus.

1901.—Dr. O. Amrein (Arosa) performed "exploration and curettage of the left iliac bone. A guinea-pig was inoculated with pus from the carious bone. Tuberculosis followed in the guinea-pig, and from these lesions tubercle bacilli were grown in almost pure culture." The sinus closed and only opened once again for a short time twelve years later.

1902.—Right sinus closed, was able to lead an active life, played tennis and hockey.

1915.—Pain in right ear. Tinnitus. High notes heard more easily than low notes. Swelling appeared over outer aspect of right orbit.

1919.—Infected tonsils removed.

1925.—Prolonged dental treatment for abscesses at apices of teeth.

1926.—Lump appeared in lower jaw and projected into right side of mouth. It was thought to be a dental cyst. Jaw became stiff.

October, 1926.—Mr. F. Barnes (Birmingham) removed portion of mass in mouth for histological section. Professor H. Wilson (Birmingham) reported, "the tumour has the structure of a 'myeloid sarcoma'. The stroma is fairly cellular and many of the giant cells are rather degenerate in appearance, but there is nothing to suggest that the growth is more malignant than is usually the case in such tumours. From the histological point of view the condition is definitely neoplastic, and the appearances cannot be explained on the basis of bone absorption or of any inflammatory process." Tumour removed from lower jaw through incision in skin.

April, 1927.—Swelling in jaw reappeared. Further operation for local recurrence of myeloma.

August.—Firm swelling in left upper jaw.

December.—Diplopia and dim vision in left eye. Professor A. Vogt (Zürich) found that "the left optic disc was definitely paler than the right. Incipient optic atrophy. The right fundus was normal. There was weakness of the left superior rectus oculi, and slight sensory loss in the area of the first branch of the left fifth cranial nerve."

January, 1928.—Could not recognize objects with left eye, but could appreciate light and dark. She was told that the tumour in the left upper jaw was a myeloma and that the defective vision was due to another myeloma. Professor P. Clairmont (Zürich) found "in the left post-nasal space arising from the left superior maxilla, a dark red swelling. It was explored and histological sections showed it to be a benign giant-celled tumour."

April.—Was told that she had optic neuritis due to pus behind left eye. Drainage of suppurative ethmoiditis by Mr. E. M. Woodman (Birmingham). Eyesight gradually improved. All bones now rather tender, so that even shaking hands was painful. Left hip very painful. Lump appeared in left thigh. Treated for rheumatism. Pain became worse. Radiogram showed large myeloma of left ilium.

June.—Following deep X-ray therapy for relief of pain she collapsed. Vomiting persisted for four days. Dr. E. M. Knott (Sutton Coldfield) called in consultation Dr. A. P. Thomson (Birmingham). Intravenous glucose was given.

December.—Recurrence of pain in left hip. At this time could hardly lift herself in bed. Deep X-ray therapy was repeated.

1929.—Other swellings appeared upon right thigh, left thigh, and lower part of right shin. These were not so painful as the previous ones.

July, 1930.—Dr. A. P. Thomson diagnosed hyperparathyroidism and confirmed this by an estimation of the serum calcium, which was found to be 13·8 mgrm. per 100 c.c.

*Past History.*—No history of rickets or tetany. Has never fractured a bone. No family history suggesting disease of bones. Catamenia commenced at age of 15; regular but rather excessive, lasting five to six days and coming on every three weeks. They ceased two and a half years ago. Had one miscarriage and then two normal pregnancies. Labours were difficult, requiring an anæsthetic and instruments. On the advice of her doctor her children were not breast-fed. Teeth were good until the age of forty, when she was found by X rays to have apical abscesses. The teeth were not affected by the pregnancies. Deafness has progressed during the last four years. One brother is very deaf, the condition dating from the age of 20. In 1929 was told she had a cataract in the left eye. Mother and two older sisters have cataracts. Always has a furred tongue. Certain articles of food cause nausea. Vomiting readily occurs after meals. No abdominal pain. No history of having passed gravel or stone. No increased frequency of micturition. Albumin has been present in the urine for some time.

Nov. 18, 1930.—Admitted to the London Hospital.

ON EXAMINATION.—Height, 5 ft. 2 in. Weight, 7 st. 8 lb. Well-nourished woman, gets about well with crutches. Hearing very poor. Better in noisy surroundings. Uses instrument for right ear. Eustachian tubes patent. Tympanic membranes normal. Weber test: louder on right side. Rinne test: bone conduction equal on both sides. Air conduction: right better than left, but much poorer than bone conduction (Mr. N. Patterson). Calvaria, normal size and contour. Sclerotics white. R.V. 6/18. L.V. less than 6/60. Extensive senile changes in the lenses of both eyes. Cataracta coronaria, water clefts, cataracta cuneiformis. Powdery cataract in concentric layers. Left primary optic atrophy, right optic disc normal (Mr. C. Goulden). Double dentures. Caries of two of six remaining teeth. Tongue slightly coated. Mucous membranes normal colour. Thyroid gland normal. No tumour felt in neck. No hypotonicity of muscles. No abnormality in nervous system. Trousseau and Chvostek signs absent. Small bony swelling on outer aspect of right orbit. Three faint scars of operation—left orbit, left superior maxilla, and right side of mandible. No abnormality of spine or bones of thorax. Scar of old sinus over centre of shaft of right humerus. No abnormality of bones of upper limbs. Operation scar on left buttock, scar of old sinus on right buttock. 2 cm. shortening of left lower limb. Slight diffuse prominence of subcutaneous surface of right tibia in its middle third. Pressure upon the bones gives rise to no pain. Heart, lungs, and abdomen normal. Blood-pressure 160/100 mm. mercury.

*Urine.*—Acid; sp. gr., 1016; albumin, 1/20 volume; Bence Jones protein absent; no sugar; deposit of epithelial cells. Blood-urea, 0·033 per cent.

*Blood-count.*—Red cells, 4,600,000; Hb., 60 per cent; C.I., 0·65; leucocytes, 5000; differential count, normal. Bleeding time and coagulation time, normal. Wassermann reaction negative.

*Radiograms of Bones.*—Cyst-like area in upper part of shaft of right femur

expanding corticalis in region of great trochanter. Similar area in left ilium adjoining acetabulum. Ankylosis and deformity of left sacro-iliac synchondrosis, evidently a change of long standing. By contrast with the bones of a control subject the right tibia and fibula (*Fig. 191*), left hand and forearm, as well as other bones, show slight but definite diminution of density. No fractures nor other deformity. Calvaria is mottled and shows three rounded pale areas in the frontal region, the largest more than 1 cm. in diameter: the tables are not thickened.



FIG. 191.—Case 4. Controlled radiogram of right tibia and fibula.

*Radiograms of Kidneys.*—Kidney shadows normal in size. Group of small calculi (largest 3 mm. diam.) seen in region of each kidney.

*Calcium and Phosphorus Metabolism before Operation.*—During the five months preceding operation the serum calcium varied between 12 and 15 mgrm. per 100 c.c., and the plasma phosphorus was only once above 2.0 mgrm. per 100 c.c. (*Fig. 192*). Plasma phosphatase 1.06 mgrm. The calcium output was estimated in the urine and faeces for one three-day period, the patient being kept on a weighed diet of known low calcium content. The calcium output in the urine was about 50 per cent greater than in the normal control, that in the faeces being almost the same as in the control (*Fig. 180*).

**FIRST OPERATION** (Mr. A. J. Walton—Nov. 26, 1930).—Under open ether anaesthesia. Collar incision made in neck. The sternomastoid muscles were retracted and the pretracheal muscles divided. Right and left superior parathyroid

bodies were identified and were normal. No tumour felt in neck on either side of trachea, even on passing the finger 3 cm. into the thorax. Inferior pole of right lobe of thyroid gland was paler and firmer than the rest of the thyroid tissue. This was evidently due to a tumour which did not, however, alter the normal contour of the thyroid gland. The thyroid gland was incised over the tumour and

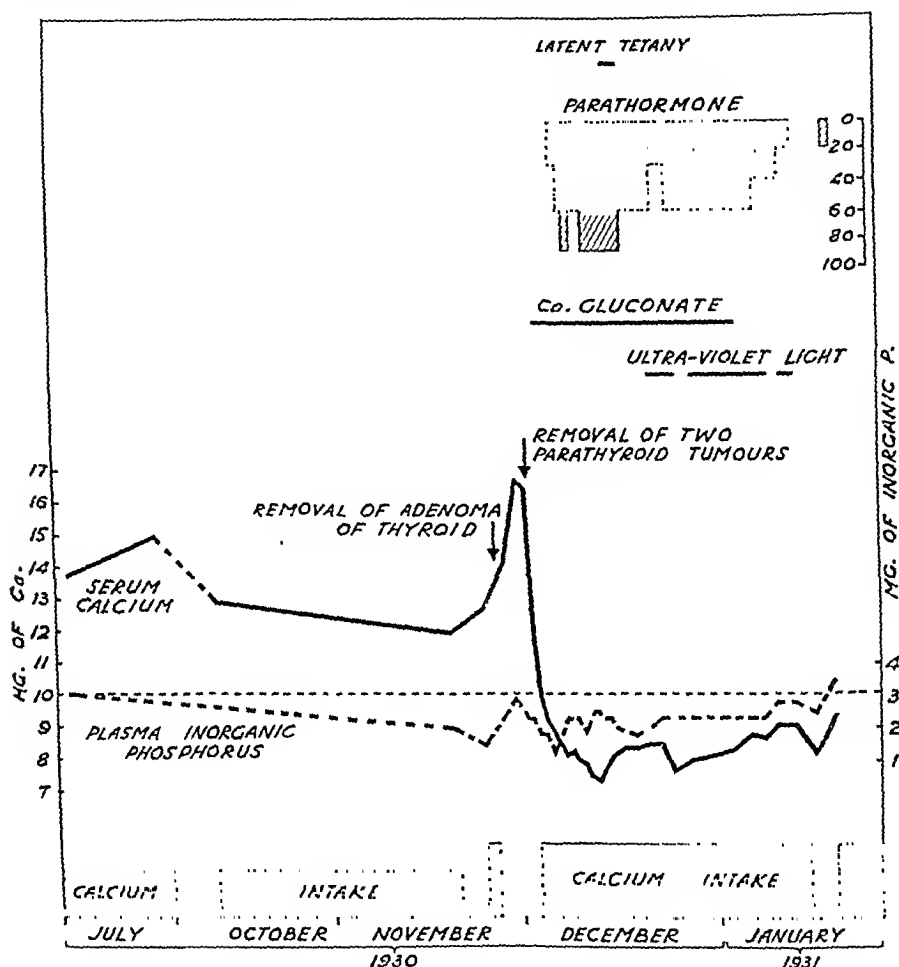


FIG. 192.—Case 4. Chemistry of the blood. The blood was examined at intervals for five months before and for two months after operation. The low calcium diet was that given during the investigation of the calcium balance. The high calcium diet was not weighed. It included four pints of milk daily, together with ice cream, cheese, and eggs. The scale indicating the dosage of parathormone represents units per day. The dosage of calcium gluconate was 10 c.c. of a 10 per cent solution daily, given by intramuscular injection.

an opaque yellowish-white swelling 2.0 by 1.5 cm. was shelled out (Fig. 193). The cut surface was glistening, firm, granular, and gritty. The thyroid gland was sutured and the wound

FIG. 193.—Case 4. Generalized osteitis fibrosa. Calcified adenoma removed at first operation from lower pole of right lobe of thyroid gland. (Actual size.)

closed. A piece of bone was removed from the corticalis of the right tibia immediately above the swelling. Chiselling was found difficult.



## Pathological Report.

(PROFESSOR H. M. TURNBULL.)

Tissues removed at first operation. (S.D. 2731/1930.)

## 1. Right Tibia.—

**MACROSCOPIC.**—A longitudinal strip of bone covered with periosteum, 5.5 cm. long, 1 cm. wide, and 0.6 cm. deep. A fresh longitudinal section through the centre showed a pearly corticalis (2 mm. deep) in which many short longitudinal red streaks were just visible to the naked eye. This merged into a gelatinous, pale yellow, rarely red, tissue traversed by some thin sheet-like trabeculae of bone.

**MICROSCOPIC** (*Fig. 194*).—Beneath the periosteum are a few bays in the bone, and some of these communicate with medullary spaces beneath. In the outer 2 mm. of the corticalis are many medullary spaces, which run for the most part in the long axis of the bone. Some of the larger intercommunicate. These spaces have evidently been formed by the widening of Haversian canals, of which very few remain. The outer contain a delicate, cellular fibrous marrow. In the inner the fibrous marrow encloses groups of adipose cells. The vessels are wide and engorged. The inner 4 mm. of the corticalis are represented by a few slender trabeculae in adipose marrow. In a few bays in these trabeculae, where there is active resorption by osteoclasts, the marrow is fibrous. There is much recent hæmorrhage in the adipose marrow, doubtless caused by the operation. The bone consists of lamellar systems enclosing several interstitial systems of woven bone. Resorption is shown in many places by single osteoclasts or groups of osteoclasts in Howship's lacunae. At the extremities of a few medullary spaces the bone has a very gnawed appearance. Active apposition is shown by numerous osteoid zones covered by large osteoblasts, and is at present in excess of active resorption. The depth of the osteoid zones is within the limits of those in healthy young children.

**Remarks.**—The compact corticalis has been extensively converted into a spongy bone of wide mesh. At the time of removal of the specimen resorption by osteoclasts was still in progress but apposition was more general. There is no evidence of osteomalacia. The appearances are those of severe osteoporosis. There is slight fibrosis of the marrow focally, and this fibrosis appears to depend upon the activity of resorption and apposition. The picture is not, however, that of frank osteitis fibrosa. The portion of bone removed for examination came from the relatively healthy part of the skeleton. It is of much greater value for the differentiation of a case of diffuse osteitis fibrosa from one of focal osteitis fibrosa than an area of severe change.

## 2. Body Removed from Lower Pole of Right Lobe of Thyroid Gland.—

**MACROSCOPIC.**—An oval body measuring  $2 \times 1.5 \times 1.1$  cm. and surrounded by a zone of thyroid gland from 1 to 5 mm. thick. On section it felt gritty. The cut surface showed numerous chalky white rounded and rosette-like nodules, closely packed in a gelatinous grey tissue. The body was sharply defined from the thyroid tissue by a very thin transparent capsule.

**MICROSCOPIC.**—Portions decalcified in nitric acid and cut upon the freezing microtome and portions decalcified in Müller's solution and embedded in paraffin showed the nodule to be an extensively calcified colloid adenoma of the thyroid gland.—H. M. T.

**PROGRESS AFTER FIRST OPERATION.**—The patient remained fairly comfortable. No tinglings in fingers. No tetany. Trousseau and Chvostek signs negative. Twenty-four hours after operation the serum calcium was 14.1 mgrm. per 100 c.c.,

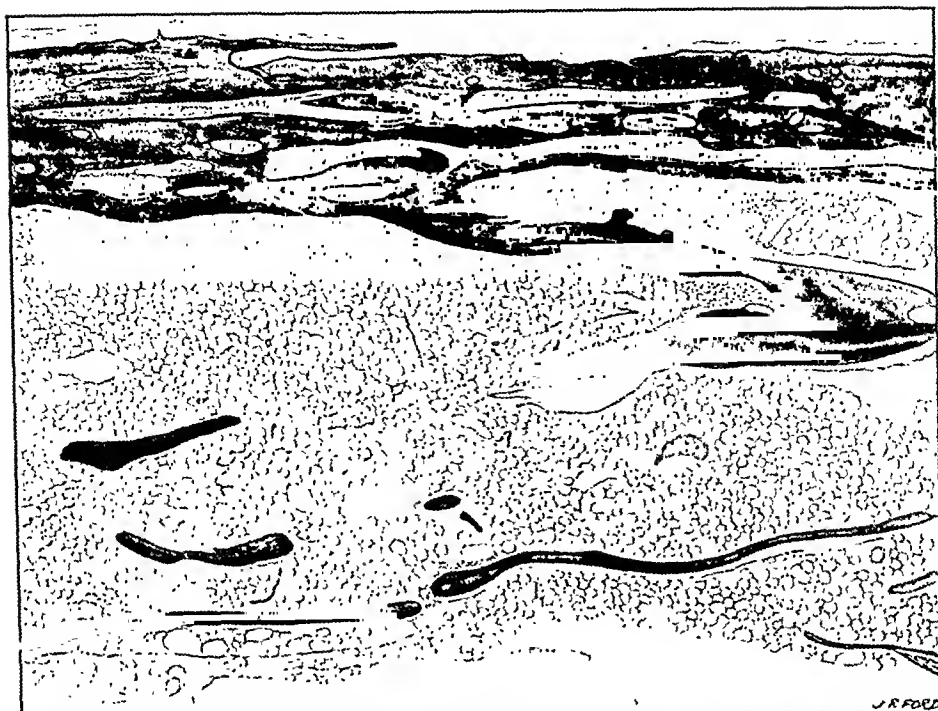


FIG. 194.—Case 4. Right tibia; corticalis. Generalized osteitis fibrosa. Note the extreme porosity; fibrosis is focal, and confined to Haversian spaces in the outer part of the corticalis. Müller's fluid followed by nitric acid; Weigert's iron-haematoxylin and van Gieson.

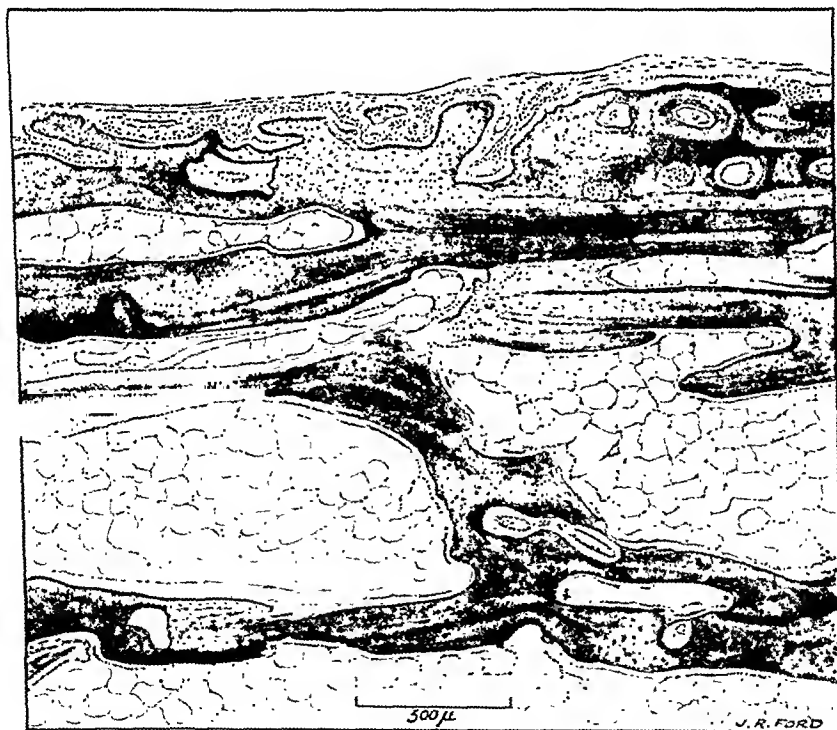


FIG. 195.—Case 9. Tibia; corticalis. Osteomalacia and osteoporosis. The osteoid zones are seen as pale seams upon the deeply stained, calcified bone. The porosity is shown by the compacta having become almost a spongiosa. Müller's fluid followed by nitric acid; Ehrlich's haematoxylin and eosin.

and the plasma phosphorus 2.3 mgrm. per 100 c.c. On the third day after operation the serum calcium was 16.7 mgrm. per 100 c.c. (*Fig. 192*). It was therefore concluded that the tumour removed was not of parathyroid origin. This view was confirmed when portions of the tumour decalcified in nitric acid and cut upon the freezing microtome showed the nodule to be an extensively calcified colloid adenoma of the thyroid.

**SECOND OPERATION** (Mr. A. J. Walton—Dec. 1, 1930).—Open ether anaesthesia. Stitches removed from old collar incision. Flaps reflected. Incision enlarged on both sides as far as upper border of thyroid cartilage. Carotid arteries exposed. Finger passed up on right and left sides as far as hyoid bone. No tumour found. Further incision along anterior border of left sternomastoid muscle to suprasternal notch. Right superior parathyroid body, measuring  $2 \times 1.3 \times 1.2$  cm. and situated deep to the œsophagus opposite the middle of the thyroid gland, was mistaken for a lymphatic gland and removed. Right inferior parathyroid body measuring  $7.5 \times 5.0 \times 1.8$  cm. was found behind trachea and œsophagus, opposite the bodies of the first and second thoracic vertebrae, well below the level of the clavicles. The inferior thyroid artery was divided and a finger inserted along the inner border of the longus colli muscle to reach the tumour. It was then displaced upwards and the pedicle to the inferior thyroid artery divided. The wound was closed in layers, and one drainage tube inserted.

### Pathological Report.

(PROFESSOR H. M. TURNBULL.)

Tissues removed at second operation. (S.D. 2778/1930.)

#### 1, 2. Right Parathyroid Bodies.—

##### MACROSCOPIC.—

1. *Larger Right Parathyroid Body* (*Fig. 196*).—The body was of elongated heart shape, with two small upward projections from the base and a bulge followed by a constriction on the left border. It measured  $7.5 \times 5 \times 1.8$  cm., and weighed 26.2 gm. It felt soft but elastic. The anterior surface was convex; the posterior was slightly concave from side to side. Portions of a thin sheet of blood-stained areolar tissue were easily removed. Between the two projections from the base a large vein lay posteriorly, and divided into a large right, large left, and a smaller median branch upon the posterior surface. Otherwise the outer surface was perfectly smooth. The posterior surface showed an area the size of a pin-head and a smaller area of opaque yellow. Cholesterol plates with notched angles were removed from these with a needle. The rest of the surface was of café au lait colour. The cut surface was soft, wet, elastic, very finely granular under a hand-lens, and of the same colour. It showed two small clefts filled with clear fluid.

2. *Smaller Right Parathyroid Body* (*Fig. 196*).—This measured  $2 \times 1.3 \times 1.2$  cm. It felt firmer than the large body and externally was of a slightly lighter yellow. On section it showed a large, round, bulging, grey-purple area (0.7 cm. in diameter) and several small purple areas in a yellow ground.

##### MICROSCOPIC.—

1. *Larger Right Parathyroid Body*.—The gland is surrounded by a thin capsule of collagenous and elastic fibres. It contains a few veins and is divided into small polygonal and rounded acini by the collagenous walls of capillaries (van Gieson's and Laidlaw's stains). In a few places the interacinar septa are wider, occasionally being hyaline but usually being either considerably or extremely rarefied by œdema. The cells are polygonal except where they border upon alveolar lumina. They are separated by a net of cell-membranes. Most of the cells measure from 10 to 12  $\mu$  and have nuclei of 5  $\mu$ . In some the cytoplasm appears dense but shows small triangular clefts and often a larger round vacuole; in most the cytoplasm consists of closely packed small granules. Scattered among these smaller cells and also occupying completely a few small areas are larger cells of 13 to 20  $\mu$  with nuclei of 5 to 6.5  $\mu$ , rarely 9  $\mu$ . Their cytoplasm consists of granules set farther

apart, and is sometimes interrupted by round or irregular vacuoles. The cytoplasm in all cells is oxyphil, and in all save the large cells is more oxyphil than is usual in pale oxyphil cells. The polygonal net of cell membranes is basophil. In sections stained by Best's method a considerable number of the cells contains a small linear or crescentic group of minute granules of glycogen in their periphery against part of the cell-membrane. A very few cells contain a larger, denser group of small granules. Larger granules of glycogen lie upon the free surface of cells lining alveolar lumina. No fatty substance is demonstrated by Herxheimer preparations except some crystals in an alveolar space. The nuclei are almost all deeply stained, but show a clear structure; in the larger examples one or more nucleoli are seen. There is a considerable number of large alveolar spaces which contain a coagulum which is usually slightly, sometimes deeply, oxyphil. The cells which bound them are chiefly of the smaller more deeply stained kinds. Tissue-mast-cells are scattered through the interacinar septa.

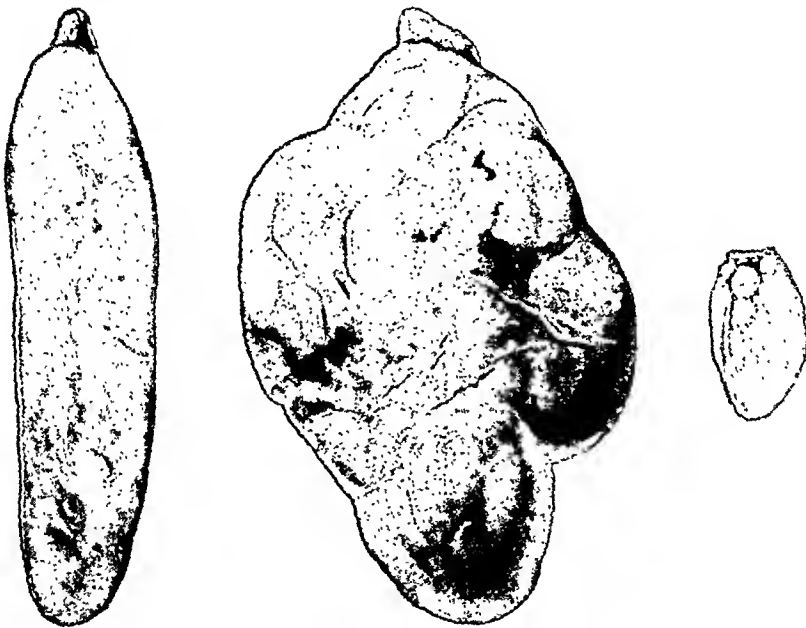


FIG. 196.—Case 4. Generalized osteitis fibrosa. Larger right parathyroid tumour and smaller right parathyroid tumour removed at second operation. (Actual size.)

2. *Smaller Right Parathyroid Body.*—The gland is divided into four large and several small lobes by septa of arcular and denser fibrous tissue. The lobes are subdivided by delicate vascular septa into acini, the size and shape of which depend essentially upon whether they contain principal cells or oxyphil cells. The principal cells occupy small rounded acini. They are usually grouped in a solid mass with cubical cells in the periphery, but frequently in one or more layers round a lumen containing oxyphil coagulum. The latter is usually thin and scanty. Some solid acini consist of a single ring of short columnar cells. Scattered oxyphil cells are found amongst the principal cells in these small acini; oxyphil cells also fill acini of similar size and shape, occasionally surrounding a lumen. Most of the oxyphil cells, however, occupy large nodular areas in which Laidlaw's stain shows the acini to be considerably larger and of less regular shape. These areas correspond to the purplish areas seen with the naked eye. The largest lobe differs from the others in being rounded, and it is occupied almost completely by oxyphil acini. It is impossible to measure the principal cells accurately because there is no visible



membrane between adjacent cells except in the case of a very few dropsical examples. The latter measure  $9\ \mu$  in mean diameter. The others have a spongy basophil cytoplasm and appear to be from 8 to  $9\ \mu$ . All have nuclei of  $5\ \mu$ . Many of the principal cells contain numerous isotropic spheres, up to  $3\ \mu$  in diameter, that are stained pale yellow in Herxheimer preparations. Where the cells surround lumina the spheres lie in the cytoplasm of the free ends of the cells. With the exception of a very few deeply oxyphil examples the oxyphil cells have a basophil membrane and resemble those in the large gland. Those that lie isolated amongst the principal cells are usually, however, smaller, measuring 8 to  $9\ \mu$  with nuclei of  $5\ \mu$ . The oxyphil cytoplasm is frequently found in large globules, some as large as red corpuscles, or forms a net of lumps of various sizes enclosing irregular vesicular spaces. These appearances are probably artificial, because frozen sections had been taken from the tissue before it was embedded, and in order to obtain them as quickly as possible the formaldehyde had been heated. The nuclei in the larger oxyphil cells are of the same sizes as in the previous section, a few giant examples reaching  $9\ \mu$ . The nuclei of both the principal and oxyphil cells are stained deeply, but show a definite structure. No preparations of this gland were made to demonstrate glycogen. The oxyphil cells occupy more than half the area of the gland.

**Remarks.**—The larger gland is occupied completely by oxyphil cells, which almost all have the structure of pale oxyphil cells although most approach the dark type in depth of eosinophilia. Many contain a little glycogen. In the smaller gland the appearances indicate that lobes are being successively converted into tumour-like masses by the replacement of principal cells in small acini by similar oxyphil cells and by the subsequent multiplication of the oxyphil cells. These appearances are strongly in favour of a functional hyperplasia as opposed to an autonomous neoplasm. The hyperplasia in this case resembles that in *Case 3* in that it consists of oxyphil cells, and almost entirely of pale oxyphil cells. It differs in that there is very much less dropsical separation of the oxyphil granules and less glycogen in the cells.—H. M. T.

**SUBSEQUENT PROGRESS.**—Before leaving the operating theatre one litre of 10 per cent glucose containing 50 units of insulin was given intravenously. On recovery from the anaesthetic the patient's voice was hoarse, and laryngoscopy revealed complete paralysis of the right vocal cord. In view of the fact that two parathyroid tumours had been removed, intramuscular injections of calcium gluconate were commenced the day after operation, 10 c.c. of a 10 per cent solution being given three times a day (*Fig. 192*). The high calcium diet was supplemented by large doses of calcium lactate, up to 15 gm. a day. A 5 per cent solution of calcium chloride was kept in readiness for intravenous use but was never required. The level of the serum calcium rapidly dropped until four days after operation it was 8.9 mgrm. per 100 c.c. The patient was restless and irritable, but there was no objective sign of tetany. At this point parathormone was given by intramuscular injection in increasing doses up to 30 units three times a day. Twelve days after operation the Chvostek sign was positive for the first time, the level of the serum calcium being 7.8 mgrm. per 100 c.c. There were no tinglings nor cramps, and the Trousseau sign was never positive. The Chvostek sign was negative on the fourteenth day after operation, and from that time no manifestation of tetany occurred. Two weeks after operation treatment by ultra-violet irradiation was commenced and carried out for four weeks. Five weeks after operation calcium gluconate injections were discontinued and a week later parathormone was given for the last time. Three weeks after operation the patient was able to walk with the aid of a stick, and four weeks later she left hospital, walking up and down stairs unaided. She pointed out that the readiness to become nauseated and to vomit had gone.

Shortly afterwards she was severely ill with bronchopneumonia, and this was followed by an abscess in the left iliac fossa from which 250 c.c. of pus were evacuated. Five months after removal of the parathyroid tumours she was again well and able to walk alone. The voice was almost normal.

*Calcium and Phosphorus Metabolism after Operation.*—As already stated, removal of the thyroid adenoma did not abolish the hypercalcaemia, whereas after removal of the parathyroid tumours the serum calcium fell abruptly. It reached its lowest point, namely 7.4 mgrm. per 100 c.c., on the eleventh day after operation (Fig. 192). By this time the plasma phosphorus level had risen to 2.5 mgrm. per 100 c.c. Subsequently the serum calcium gradually rose, until 7 weeks after operation it was 9.4 mgrm. per 100 c.c., although all treatment except a high calcium diet had ceased. Plasma phosphatase 0.635 mgrm. Six weeks after operation the calcium output in urine and faeces was estimated for one three-day period on the same low calcium diet as before. The figures both for urine and for faeces were as low as those found in cases of post-operative tetany (Fig. 180).

**Remarks.**—It is worthy of note that a parathyroid tumour measuring 7.5 cm. in its greatest dimension can be so deeply situated in the neck as to defy palpation. There are certain cases recorded in the literature (Barrenseheer and Gold, 1928; Bauer, Albright, and Aub, 1930; Wendel, 1930, *Case 2*) where the evidence of hyperparathyroidism is conclusive and yet no parathyroid tumour was discovered at operation. In recording his case Ball (1930) discusses the possibility that the tumour was missed at operation. In all these cases it would seem imperative to explore the neck again. Removal at operation of two parathyroid tumours has been recorded previously (Beek, 1928). In this case post-operative tetany occurred on the fifth day, accompanied by a psychosis. The patient died of exhaustion on the twentieth day. In such circumstances intravenous calcium chloride must be used. The size of the tumour evidently bears no relation to the severity of the bone lesions, for in *Case 2* the patient was much more severely crippled than in *Case 4* and yet the tumour measured only  $2.3 \times 1.5 \times 0.9$  cm. and weighed only 1.3 gm.

Epigastric pain, anorexia, and nausea have been described in hyperparathyroidism (Ball, 1930). The rapid disappearance of vomiting after removal of a parathyroid tumour has also been recorded (Pemberton and Geddie, 1930; Rosenbach and Disqué, 1930).

### CLINICAL CHARACTERISTICS OF HYPERPARATHYROIDISM.

In the following description are included the cases from the literature summarized above and also the four cases described at length in this article. It will be seen that the symptomatology of hyperparathyroidism does not rest with the skeleton alone.

Satisfactory proof of hyperparathyroidism now exists in 32 cases of generalized osteitis fibrosa. In 21 of these a parathyroid tumour was removed by operation, in 2 cases two parathyroid tumours were removed, and in 4 cases no parathyroid tumour was found at operation. The remaining 5 cases were not operated upon, but proof was obtained by autopsy of a parathyroid tumour in 3 cases, and of two parathyroid tumours in 1 case. The condition is reported twice as frequently in women as in men, the commonest ages being 30 and 55. Pain and tenderness in the bones were common, and

osteoclastomata occurred in more than half the cases. Spontaneous fractures were recorded in 14 cases. Thirst and polyuria occurred in 5 cases, renal calculi in 10 cases, and hæmaturia in 1 case. Occasionally renal colic was the presenting symptom. Hypotonicity of muscles was described in 7 cases. In only 6 cases was the parathyroid tumour palpable in the neck. The largest tumour removed measured  $7.5 \times 5.0 \times 1.8$  cm. and weighed 26.2 grm., and yet it had not been palpable in the neck owing to its situation behind the trachea. The size of the tumour bears no relation to the severity of the bone lesions, for in the most severely crippled patient the tumour measured  $2.3 \times 1.5 \times 0.9$  cm. and weighed only 1.3 grm. Nausea and vomiting, sometimes with abdominal cramps, have been described. Wasting is common in advanced cases. In those cases where radiograms are mentioned there was greatly diminished density of bone shadows. This is best demonstrated by the controlled method. Sometimes there was radiological evidence of cysts, but these only rarely expanded the corticalis. The serum calcium figures varied between 12.6 and 23.6 mgrm. per 100 c.c., the plasma phosphorus between 1.0 and 2.7 mgrm. Where the plasma phosphatase was estimated it was invariably high. The output of calcium in the urine varied from a slight increase to eight times the normal figure.

In the majority of cases pain in the bones was abolished immediately after operation, and the mechanism of this is difficult to conceive. Decrease in size of osteoclastomata within a few weeks after operation has been twice recorded. Restoration to normal of the menstrual function has also been observed. In three cases removal of the tumour promptly abolished polydipsia and polyuria. In one case renal calculi began to break up a few weeks after operation and were passed into the bladder. In many cases the levels of the serum calcium and plasma phosphorus and the excretion of calcium in the urine were restored to normal. Sometimes there was temporary hypocalcæmia and even oliguria, with subnormal excretion of calcium in the urine. In all five cases where it was recorded after operation the plasma phosphatase remained high; in one of these it showed no alteration even seventeen months later. Latent tetany after operation was common, and manifest tetany often occurred, with marked variations in severity. It was fatal in one case only, where two parathyroid tumours were removed. Nevertheless removal of two parathyroid tumours is not necessarily fatal, nor even necessarily accompanied by post-operative tetany. In one case tremor and acute mania occurred three weeks after operation, at a time when the serum calcium had dropped to 6.6 mgrm. per 100 c.c. The condition was completely relieved in three days by the use of parathormone injections in conjunction with a preparation of calcium given intravenously. This latter procedure must undoubtedly be regarded as the essential treatment of severe post-operative tetany following removal of a parathyroid tumour. The post-operative treatment should include a high calcium diet and ultra-violet irradiation as routine procedures.

Symptomatic improvement almost always occurs after removal of a tumour. Not only is pain in the bones abolished, but there is marked gain in weight, and many crippled patients have been able to dispense with sticks and crutches. In one case, however, pain in the bones was not entirely

abolished eighteen months after operation. Where no parathyroid tumour was discovered removal of a normal parathyroid gland had no good effect in two cases, but removal of two normal parathyroid glands led to considerable symptomatic improvement in another. Some authors claim that an increase in density in the radiographic shadows of bones was to be seen in their cases a few months after operation. It is of great interest that three and a half years after operation Mandl could demonstrate no such change in his case. Should there be special reasons contra-indicating operation upon the neck, or should no tumour be found, the patient may be treated with irradiated ergosterol in the hope of increasing the apposition of bone. In at least one case good evidence has been obtained of the effect of such treatment in increasing the density of bone as judged by radiograms.

### OPERATIVE TECHNIQUE.

Mr. A. J. Walton has kindly made the following note on the technique he employs at operation.

A wide exposure is essential, for not only is it necessary to explore all the parathyroid glands, but also, as our limited experience has already shown, it is sometimes necessary to search behind the trachea and in the mediastinum. In all our cases a wide collar incision has been made, the sternomastoids freed and widely retracted, and the pretracheal muscles between them divided transversely in one sheet at the level of the centre of the isthmus of the thyroid gland. The flaps thus formed are dissected up as high as the upper poles of the lateral lobes and as low as the sternum. This gives a much wider exposure than a vertical split of the muscles, and if they are carefully sutured afterwards no deformity or weakness results.

The lateral lobes of the thyroid gland are now rolled inwards in turn to expose the sites of the normal parathyroid glands. These are examined in turn, and a tumour may at once be found in the normal site or a little lower. In a case published elsewhere (Hunter, 1929; 1930, *Case 4*) a tumour measuring  $3.7 \times 3$  cm. was at once found at the site of or a little lower than the left inferior gland. If no tumour be observed in the usual site, a finger should be passed into the thorax on either side of the trachea, for the tumour may pass down in this situation. In *Case 3* a tumour  $6.8 \times 2.8$  cm. was easily detected in the upper part of the thorax on the left side of the trachea and was readily drawn up into the neck. In certain cases no tumour is visible. A difficulty may arise if there be a small thyroid adenoma, for a parathyroid tumour may be situated in the substance of the thyroid gland. In *Case 4* this error was made in spite of the fact that Professor Turnbull, who was present, was of the opinion that the tumour was a colloidal adenoma of the thyroid gland. This error should be guarded against, because the yellowish-brown appearance of a parathyroid tumour usually makes it distinctive even to the naked eye. In our case the error was only made clear after histological examination, and a second operation revealed a parathyroid tumour behind the trachea. In *Case 2* also the tumour lay behind the trachea. This anatomical relationship probably explains many of the reported cases where the tumour has not been found. If, therefore, a tumour is not recognized in

one of the ordinary positions nor in the thorax at the side of the trachea, the right thyroid lobe should be rolled over to the left and the carotid sheath retracted outwards until the right inferior thyroid artery is visible as a band passing to the right lobe. A small incision is then made into the fascia above it and just *behind* the œsophagus. The prevertebral space is thus entered and a small tumour may be immediately revealed as in *Case 2*. If it is not, a finger can be inserted into the space and passed down behind the œsophagus into the thorax. At the second operation on *Case 4* this was done and a soft fleshy mass measuring  $7.5 \times 5$  cm. was easily felt lying on the first and second thoracic vertebræ. With a few movements of the finger it was freed from its adhesions and pulled up into the neck, when it was found to have a narrow vascular pedicle attached to the inferior thyroid artery. This was divided and the tumour removed. In the case of a tumour arising on the left side it would be necessary to make an incision through the fascia on the left side. The tumour could then be everted and the pedicle ligatured. The cavity after removal appears to be of remarkable depth, but in our case the elevation of the tumour was accomplished with extraordinary ease. A tube is inserted into the cavity, the pretracheal muscles sutured with catgut, and the skin incision closed. We have usually left the tube in for two or three days.

### PATHOGENESIS OF GENERALIZED OSTEITIS FIBROSA.

(PROFESSOR H. M. TURNBULL.)

Generalized osteitis fibrosa is characterized by changes in the skeleton, enlargement of one or more parathyroid glands, hypercalcæmia, and an increased excretion of calcium in the urine.

The changes in the skeleton are lacunar resorption, apposition, fibrosis of the marrow, and the formation of osteoclastomata and cysts. Lacunar resorption is the predominant change. Radiography, and histological examination of the skeleton at necropsy show that resorption has led to a general osteoporosis, which, however, varies in degree in different places. But formation of bone does not cease. Bone is seen in process of formation in almost all sections taken from the more severely affected parts of the skeleton, and previous formation of bone is shown by an extensive, and in places complete, replacement of lamellar bone by trabeculae of woven bone. Apposition is, indeed, frequently the predominant active process in a segment examined. The combination of resorption and apposition is shown by osteoclastic and osteoblastic activity upon different trabeculae in many sections, and even upon the same trabecula in some sections. The interplay of the two processes is further witnessed by trabeculae that are composed of several separate systems of woven bone. In focal areas the new spongy bone that has been formed may actually exceed in bulk the normal spongiosa and compakta, for instance in some of the expansions of the ribs in a case previously reported (Hunter, 1930, *Case 3*) and in the massive calvaria of *Case 1*.

Where apposition is taking place the depth of the osteoid zones varies in different places in the same case, and the maximal depth varies in different cases. The essential abnormality in rickets and osteomalacia is cessation of

calcification, and the characteristic histological abnormality that results is an excessive amount of osteoid tissue. Consequently, the disputed question of whether or not there is a true osteomalacia in generalized osteitis fibrosa depends upon whether the amount of osteoid tissue is, or is not, excessive. Pommer (1885) showed that all bone when first formed is osteoid and becomes calcified later. Osteoid zones are, therefore, present upon all trabeculae during osteoblastic activity. The depth of the zones varies with the rapidity of formation. Thus, the depth is greater in the rapidly growing bones of children than in the bones of adults, and is greater still in the healing of fractures. In order to determine whether there is an abnormal delay in calcification it is necessary first to determine whether the osteoid zones are associated with osteoblastic activity and to form some estimation of that activity, and secondly to compare their depth with zones measured in healthy subjects. Some estimation of the activity is obtained from the form and number of the osteoblasts. The osteoblasts are cells of the fibrous reticulum of the marrow in a stage of functional activity. The resting cells in the fibrous marrow are spindle cells with very scanty cytoplasm. The active osteoblasts upon the osteoid zone are plumper and have a much more abundant, finely granular, more oxyphil cytoplasm. In very rapidly growing bones one or two rows of cells that show transitional characters are sometimes seen between the row of osteoblasts upon the osteoid zone and the resting cells of the marrow. Further, the active osteoblasts are more numerous, being at a shorter distance from one another, than the resting cells. When apposition ceases the osteoblasts return to the form and number of resting cells. The importance of forming an estimation of activity is illustrated by the example in this paper of a condition other than osteitis fibrosa (*Case 9*). Many of the osteoid zones were deeper than in healthy adults and children, but the depth seldom exceeded that in healing fractures, and the amount of osteoid tissue was very slight (*see Fig. 195*) compared with that in typical cases of osteomalacia. The osteoblasts, however, appeared to be in a state of rest or of very slight activity. The unusual depth could not, therefore, be attributed to exceptional activity of apposition, but indicated delay in calcification. Disturbance of calcification was further indicated by partial calcification within some zones. The histological diagnosis of osteomalacia was confirmed by the effects of appropriate treatment. In measuring the depth of osteoid zones it is necessary to select zones that have been cut strictly at right angles to their surface. In such sections the osteoblasts are at their narrowest and of spindle shape. When the cells are seen in full-face the nuclei are rounded, the cytoplasm is much more abundant, and the outline polygonal. In oblique sections the appearances approximate to those of cells seen in full-face. In a skeleton fully examined (Hunter, 1930, *Case 3*) and in *Case 1* and *Case 4* of this paper it is possible to examine portions of bone that are free from fibrosis. In *Case 3*, 1930, and *Case 1* such trabeculae are almost everywhere free from osteoblastic activity, and are fully calcified; where there is any osteoblastic activity it is relatively slight and the osteoid zones are very narrow. In *Case 4* there are many osteoid zones upon trabeculae that are not involved in fibrosis; the depth is within the limits of those in healthy children, and osteoblastic activity is as great as in children. When the trabeculae within

the fibrous marrow are considered, in *Cases 1* and *4* the zones are never deeper than in the bones of young children, in the skeleton fully examined they are seldom deeper, in *Cases 2* and *3* and *Case 4* of a previous paper (Hunter, 1930) they are usually deeper. In the four cases, however, in which the maximal depth exceeds that in young children, it is within the limits measured in healing fractures in healthy adults, and osteoblastic activity is as great as in healing fractures. Examination of these six cases, therefore, of generalized osteitis fibrosa associated with parathyroid enlargement shows that in the fibrotic areas apposition is frequently as rapid as in the healing of fractures and the osteoid zones are correspondingly deep, but neither here nor in the available parts of the skeleton that are free from fibrosis is there evidence of the delay in calcification which characterizes osteomalacia and rickets.

Schmorl (1926) said that the question of the presence of osteomalacia in generalized osteitis fibrosa cannot be decided by examination of the diseased portions of bone involved in fibrosis, because there is always active apposition there and abundant osteoid tissue is consequently to be expected. He examined, therefore, the 'healthy' parts of the bone in 44 cases. In 8 he found evidence of osteomalacia, in that osteoid zones were present, which certainly were shallower than those in his very numerous examples of osteomalacia but yet were found everywhere. The comparative shallowness he attributed to the presence of a very considerable atrophy in the bones. He made no mention of the condition of osteoblastic activity upon these zones. In the remaining 36 cases there were no calcium-free zones in the parts free from fibrosis. He said, therefore, that Lang (1926), who considered that generalized osteitis fibrosa is always combined with osteomalacia or late rickets, and Stenholm (1924), who came to an exactly opposite conclusion, were both wrong. He concluded, however, that generalized osteitis fibrosa is an independent, peculiar disease of the skeleton which has no relation to *florid* rickets or *florid* osteomalacia. Schmorl evidently in this paper made no distinction between the multiple focal areas of osteitis fibrosa and the generalized osteitis fibrosa which Hunter (1930) has differentiated by both radiographic examination and estimation of calcium metabolism. In the discussion following the review of osteitis fibrosa by Christeller (1926) in the same year, Schmorl said that he had examined the parathyroid glands in all his cases of osteitis fibrosa and had found enlargement in only four cases. In these four cases there was a combination with osteomalacia. Apparently, therefore, Schmorl found the above evidence of osteomalacia in all his four cases of generalized osteitis fibrosa associated with parathyroid enlargement, and in four out of forty cases of osteitis fibrosa without parathyroid enlargement. Schmorl (1926) described the frequency of rickets and osteomalacia in Dresden as enormous. Osteomalacia might well, therefore, be an accidental complication of generalized osteitis fibrosa in his material.

Fibrosis of the marrow is a conspicuous feature. It is very widespread, but is not universal. As in *Case 4* there may be great osteoporosis whilst fibrosis is focal and slight. In an extensive examination of a whole skeleton, however, there was no section in which fibrosis was completely absent. Where it is most sparse it is confined to bays eroded upon the surfaces of the compacta or trabeculae in the spongiosa, and to Haversian spaces eroded within the compacta or within the trabeculae of the spongiosa. In greater amount it forms a zone upon one surface of greatly eroded trabeculae in the spongiosa or forms a sheath all round such trabeculae. In still greater amount it forms broad bands in the spongiosa that enclose groups of small or slender trabeculae. All these grades are frequently seen in a single section, as is

shown in the head of the humerus in *Case 1* and very conspicuously in the bodies of the vertebrae in a case previously reported (Hunter, 1930, *Case 3*). In part of the bodies of these vertebrae trabeculae of lamellar bone form a spongiosa of normal mesh and show only focal areas of fibrosis that fill bays eroded upon their outer surfaces or fill Haversian spaces eroded within them; other trabeculae of lamellar bone are centrally split to a greater or less extent by eroded spaces filled with fibrosis; others are split into smaller pieces which are completely embedded in fibrosis; finally, areas are occupied by a close network of broad bands of fibrosis containing very closely packed small trabeculae of newly formed, woven bone. In its greatest amount the fibrosis forms in portions of the long bones that normally have a medullary cavity free from bony trabeculae a fibrous compacta containing small bony trabeculae, and in spongy bones or spongy portions of bones fully occupies both the compacta and spongiosa. In the former case the intimate relation of the fibrosis to the bony skeleton is most striking (*Case 1, Fig. 176*). In the latter case there may be focal expansions, particularly of the ribs. Where the fibrosis forms broad bands in the spongiosa or extends through the corticalis, the included trabeculae are sometimes (*Case 3*) remnants of old bone, but usually they consist of woven bone formed during the disease. Where the fibrosis extends throughout both corticalis and spongiosa—for instance, in expansions—trabeculae of woven bone may extend throughout, forming an osteosclerotic nodule; but usually they are confined to the periphery, leaving in the centre a fibrous area of variable size. The smaller focal areas of fibrosis are almost invariably associated with very active lacunar resorption, shown by numerous large osteoclasts, but sometimes with an active apposition. More extensive fibrosis is associated with exceptional activity of either resorption or apposition, or with evidence of very great previous resorption. On the other hand, where fibrosis is absent osteoporosis may be great and bear witness to much previous resorption, but the osteogenetic marrow is almost invariably at rest; occasionally it shows a slight osteoblastic activity.

Study of the fibrosis of the marrow thus shows first that it is intimately associated in position with bone, appearing first upon its surface and later ensheathing or largely replacing it, secondly that it is closely associated with exceptional activity of resorption or apposition. In infections the osteogenetic fibrous tissue of the marrow reacts like other soft tissues, and when the resulting granulation tissue is little differentiated and cellular it tends to cause resorption of bone, and when it is more differentiated and fibrous it tends to form bone. The fibrosis of the marrow and the resorption and apposition of bone in generalized osteitis fibrosa might, therefore, be the result of a spreading inflammation of the marrow. In that case the intimate association of the fibrosis with bone could only be explained by the excitement of the inflammation being present within bone. There is no histological evidence of this: the fibrosis appears initially upon the surface of portions of bone that appear to be perfectly normal in structure. Cellular proliferation of the osteogenetic marrow is excited, however, not only by inflammatory stimuli but by the stimuli that govern normal apposition and resorption of bone. In resorption and in apposition, even of lamellar bone, there appears always to be some proliferation of the cells of the osteogenetic, as opposed



to the hæmatogenous, marrow. In the more actively growing portions of the bones of the newly born a proliferation of spindle cells immediately behind the ripe osteoblasts upon the trabeculæ sometimes gives the appearance of a zone of slight fibrosis upon the trabeculæ. In severe rickets and osteomalacia focal areas of fibrosis may lie upon trabeculæ, zones of fibrosis may ensheath trabeculæ, or there may be a more extensive fibrosis. The fibrosis resembles that in generalized osteitis fibrosa, and has the same intimate connection with bone. In severe rickets the weakening of the bone following arrest of calcification leads in places to the formation of osteoid tissue in excess of the amount of bone in a normal skeleton, as is shown by the excessive width of the osteoid trabeculæ and by the buttressing of bowed corticalis. Osteoblasts are numerous and large upon the osteoid zones. There can be little doubt that the osteogenetic marrow is excited here to exceptional activity by stimuli of strain and stress. The associated zones of fibrosis are doubtless caused by the same stimuli, and are probably merely expressions of marrow in a condition of exceptional osteogenetic activity. If such fibrosis of the marrow is merely a sign of exceptional activity, and if it can be shown that the enlarged parathyroid glands in generalized osteitis fibrosa are stimulating an excessive lacunar resorption of bone, then the fibrosis in this disease can be attributed primarily to excessive stimulation of resorption by parathyroid secretion and secondarily to excessive stimulation of apposition by strain and stress upon bone weakened by great resorption.

The engorgement that is usually present in the fibrous marrow can be attributed to functional activity of the osteogenetic marrow. Thus, engorgement is associated with activity of the hæmatogenous marrow, as is shown conspicuously in the small focal areas in adipose marrow which mark the beginning of an extension of blood-formation. Red marrow, indeed, owes its colour to this engorgement.

Cysts are usually numerous. They were very numerous in one case previously reported (Hunter, 1930, *Case 3*), and the following remarks upon their formation are based chiefly upon a study of this skeleton. Many, particularly the larger, have a wall of dense, often hyaline, collagenous fibrous tissue. In the others the wall does not differ from the surrounding fibrous marrow. The dense wall appears to be a secondary change, and the cysts that have no specialized wall appear to represent earlier stages in formation. Most of the latter contain a thin albuminous coagulum with or without red corpuscles, others contain a similar coagulum with fat-granule-cells and, rarely, cholesterol crystals; others contain blood or a little fibrinous clot containing red corpuscles; others are empty. Some have a sharply defined, even margin and are lined with a layer of flattened cells that resembles endothelium. In others the margin is not sharply defined, and is obviously formed by the cells of the surrounding tissue, sometimes including osteoclasts. Occasionally the boundary consists of fibrous marrow split into a loose reticulum by hæmorrhage. The cysts most frequently occur in the larger areas of fibrous marrow that are free from bone, but they are not confined to these. Thus, the cyst in the calvaria of *Case 1* lies in a close net of bony trabeculæ. In the centres of these larger areas of fibrosis vessels are scanty or absent, and there is usually degeneration. The commonest

degeneration is a fibrinoid degeneration preceded by extravasation of red corpuscles, but there may be severe hæmorrhage, or multiple focal areas of serous œdema, or, rarely, a severer degeneration and necrosis associated with the accumulation of fat-granule-cells and cholesterol crystals. Organization of thrombosed vessels is also seen. In the periphery of the larger areas of fibrosis there are often dilated vascular spaces, of which some are distended with blood but many are empty or contain a few red corpuscles or a little thin albuminous coagulum. Such dilated vascular spaces are often present in the immediate neighbourhood of cysts. The cysts that have a sharply defined margin lined with a layer of flattened cells resemble these dilated spaces so closely that an origin from them is strongly suggested. Laidlaw's silver impregnation shows, however, that the vascular spaces have a sharply defined wall, which consists at least of a thin sheet of delicate fibrils; and the early cysts rarely have a definite and continuous limiting membrane. It is rare, too, for the cysts without densely fibrotic walls to have a lining that resembles endothelium, although large cysts with dense walls frequently do. Further, transitions can be followed from irregular areas of focal serous œdema in which the cells of the fibrous marrow are slightly separated by thin coagulum, through rounded areas in which spindle fibroblasts form an ever scantier net in such coagulum, to perfectly spherical cysts that contain coagulum alone. Again, in the neighbourhood of cysts transitions can sometimes be followed from groups of fat-granule-cells to irregular or rounded cystic spaces more or less filled with a granular debris containing fat-granule-cells and cholesterol crystals. If the cysts have a single origin, then the evidence appears to be stronger for origin in œdema and degenerations of the fibrous marrow than in dilatation of vascular spaces. It is possible, however, that cysts arise in both ways. In either case there can be little doubt that the cysts are due to disturbances in the circulation consequent upon the fibrosis of the marrow or the weakening of the bone. It is probable that cysts are also occasionally the result of autolysis of osteoclastomata. Large, ill-defined, cystic spaces, filled with thin coagulum, certainly do occur within osteoclastomata.

The osteoclastomata are areas occupied by numerous large osteoclasts among fibrocytes that are less differentiated than in the remainder of the fibrous marrow. In number and size the osteoclastomata vary greatly in different cases. Some are minute (*Fig. 187*); others form large masses that expand part (*Cases 2 and 3*), or the whole, of the circumference of large long bones. It has been pointed out (*Case 2*) that it is impossible to attribute such tumours to collections of osteoclasts that have persisted *in situ* after eroding bone. Areas of fibrinoid degeneration are preceded by extravasation of red corpuscles, and osteoclasts are usually numerous in such areas. Further, osteoclastomata are frequently associated with hæmorrhage and the accumulation of iron-pigment. Again, some of the osteoclasts occasionally contain red corpuscles or hæmosiderin. But hæmorrhage may be trivial or absent within osteoclastomata, and the number of osteoclasts that do contain corpuscles or pigment is relatively minute. It is impossible, therefore, to regard the osteoclastomata as areas in which osteoclasts have been stimulated to multiply as scavengers of hæmorrhage. Osteoclastomata have been

observed to disappear after excision of enlarged parathyroid glands (Barr, Bulger, and Dixon, 1929; Hunter, 1931). Their disappearance after excision of enlarged parathyroid glands suggests that they are areas of osteogenetic marrow that have been stimulated by excessive parathyroid secretion to an exaggerated osteoclastic activity. It has been argued that the fibrosis of the marrow represents osteogenetic marrow in an exceptional state of activity, and that this activity might be the result of excessive parathyroid secretion. In their origin the osteoclastomata would thus be akin to the fibrosis of the marrow, and the less differentiated form of the fibroblasts within them is in keeping with a more exaggerated activity than in the rest of the fibrous marrow.

According to the above interpretation of the histological changes in generalized osteitis fibrosa in man all the abnormalities in the bone could be attributed primarily to parathyroid secretion causing an excessive stimulation of osteoclastic resorption. Clinical observations bring evidence that the enlarged parathyroid glands deprive the skeleton of calcium. The hypercalcaemia that characterizes the disease disappears almost immediately after the removal of the enlarged glands, and the skeleton becomes stronger. Further, the administration to man of parathormone brings into the blood not only calcium, but lead (Hunter and Aub, 1926), radium (Flinn and Seidlin, 1929), and other elements that are known to be stored in the bones in close association with calcium. To prove that the bone of the skeleton is reduced in this process, and is reduced by osteoclastic resorption, requires animal experiment. Bauer, Aub, and Albright (1929) found naked-eye evidence of loss of trabeculae in the spongiosa of the long bones of rabbits which had been injected with increasing doses of parathormone. They made no microscopic examination, however, so that if their experiments proved that parathormone reduces the skeleton, the method of reduction was not determined. In order to supply this important deficiency Dr. F. B. Byrom in May, 1930, made a small series of similar experiments under Dr. Hunter's direction. After handing the bones to me Dr. Byrom went to America. It was intended to allow the bones to decalcify in Müller's solution until his return, but when this article was being written as many of the bones were embedded and cut as time permitted.

Dr. Byrom chose a litter of seven rabbits, two months old. All the rabbits received an unrestricted diet of bran, oats, lettuce, and cabbage. Four rabbits (316, 317, 318, 319) were given daily subcutaneous injections of parathormone (Collip) in increasing doses, beginning with 1 unit and rising to a maximum of 50 units in one animal (316). The three others (313, 314, 315) were kept as controls on the other side of the animal house. All rabbits were marked by a stamped metal ribbon clipped round a leg. No symptoms of hypercalcaemia were observed. The injections were begun on May 6. *Rabbit 317* was killed on June 16, having received in the 41 days 37.4 units in all; control rabbit 314 was killed at the same time. The serum calcium of rabbit 317 was 12.9 mgrm. per 100 c.c. on May 6 before the first injection, 9.1 on June 4, and 7.7 on June 10. The plasma phosphatase of the control was 0.315 on June 4. *Rabbit 318* was killed on July 23, having received in the 78 days 610 units; control rabbit 313 was killed at the same time. The serum calcium was 13.4 on May 6, 11.6 on May 16, and 10.8 on June 24; the plasma phosphorus was 7.0 mgrm. on June 24. In the control on June 24 the serum calcium was 9.2 and the plasma phosphorus 7.5; on June 4 the plasma phosphatase

was 0.369. Rabbits 319 and 316 were both killed on Aug. 15 with control rabbit 315. Rabbit 319 had received 770 units in the 101 days. The serum calcium was 13.8 on May 6 before the first injection, 12.0 on May 12, 10.3 on June 24, 9.8 on July 17, and 11.3 on Aug. 13; the plasma phosphorus was 7.5 on June 24; the plasma phosphatase was 0.192 on July 17, and 0.172 on Aug. 13. Rabbit 316 had received 2150 units in the 101 days. The serum calcium was 14.5 on May 6 before the first injection, 13.1 on May 12, 12.5 on May 26, 8.4 on June 24, 9.7 on July 17, and 9.5 on Aug. 13; the plasma phosphorus was 5.8 on June 24; the plasma phosphatase was 0.199 on July 17, and 0.147 on Aug. 13. In the control rabbit the serum calcium was 9.0 on June 24, and 9.7 on Aug. 13; the plasma phosphorus was 6.0 on June 24; the plasma phosphatase was 0.294 on May 26, and 0.286 on Aug. 13.

When the rabbits had been killed the long bones were dissected out, and each bone from the injected animal was radiographed beside the corresponding bone of the control. The bones of both animals were then divided longitudinally; one half was digested in pancreatic solution, the other was reserved for histological examination. When the bones were examined with the naked eye the zone of proliferating cartilage in rabbit 317 was seen to be narrower and less translucent than in the control, whilst the zone of spongiosa beneath was narrower. In rabbit 318 the zone of proliferating cartilage did not differ from that in the control; but the depth of the spongiosa beneath was strikingly less, as is well shown in photographs of the macerated bones. It appeared, therefore, that in spite of the fall in the serum calcium the experiments showed in the injected rabbits a depletion of the reserve of calcium in the bones such as had been observed by Bauer, Aub, and Albright.

**Microscopic Examination.**—In all the bones examined the provisional calcification of cartilage is complete and there is no irregularity in chondral ossification, whilst the zones of osteoid tissue are covered by closely packed, large osteoblasts, and where they are cut at right angles to their surface, their depth shows little variation and lies well within the limits of the zones in young children. There is, therefore, no evidence of rickets or osteomalacia.

**Rabbit 317 (374 Units of Parathormone) and Control.**—Two ribs and a piece of parieto-occipital bone, decalcified in Müller's solution, have been examined in each case. In the ribs in both cases there is no obvious abnormality in the spaces between the trabeculae of calcified cartilage upon which little or no bone has been deposited; the spaces contain a few osteoclasts and numerous osteoblasts at the sides of the central capillaries. In the scanty spongiosa which forms the rest of the metaphysis there are numerous osteoclasts in Howship's lacunae, and where these are absent osteoblasts usually lie upon osteoid zones. Osteoclasts frequently lie upon the truncated diaphysial ends of the trabeculae. The trabeculae are surrounded by a zone of delicate fibrosis. The corticallis for seven millimetres is reduced to a narrow spongiosa by cavities formed by erosion from the periosteal and endosteal surfaces and by erosion about vascular canals. The cavities are filled with fibrous marrow, and are lined by osteoclasts or by osteoid zones covered by osteoblasts. The inner surface of the corticallis is covered by a zone of fibrosis which unites with the fibrosis in the spongiosa of the metaphysis. Farther away from the epiphysis erosion of the corticallis is less, but is shown by many large Haversian spaces filled with fibrous marrow and lined with osteoclasts, and by deep or long shallow bays eroded from the endosteal surface. The zone of fibrosis upon the inner surface of the corticallis is continued; it varies in depth according to the amount of erosion, and is absent in a few places where there is no evidence of erosion. Active apposition by osteoblasts is present in addition to conspicuous osteoclastic resorption. The marrow within the medulla is bounded above by the fibrosis in the spongiosa and laterally by the zone of fibrosis upon the inner surface of the corticallis. It is an adipose marrow loaded with healthy hæmatogenous cells; it contains no bone and shows no trace of fibrosis. In the occipito-parietal bones the inner and outer tables contain cavities eroded from their surfaces, especially the inner, and cavities eroded round vessels. The tables are frequently interrupted. The trabeculae in the scanty spongiosa also show erosion. The cavities within the tables are filled with fibrous

marrow, and a zone of fibrosis clothes the inner surfaces of the tables and the trabeculae wherever they show erosion. Osteoclasts in lacunae and osteoblasts upon osteoid zones are numerous.

In the ribs of rabbit 317 the trabeculae of the spongiosa are less numerous than in the control and extend for a shorter distance towards the medullary cavity. Growth in length of the ribs is much less active, as is shown by a shallower zone of cartilage cells in columns, and very much shallower zones of hypertrophied cartilage cells and of calcified chondral trabeculae with little or no bone upon their surfaces. In the calvaria there is no appreciable difference between the injected rabbit and the control.

*Rabbit 318 (610 Units of Parathormone) and Control.*—As macroscopic differences were most obvious in this pair all the bones in process of decalcification in Müller's solution were further decalcified in nitric acid, embedded, and cut. The bones were: portions of two ribs and of the parietal bone; the whole length of the humerus, femur, tibia, and first metatarsal; the tibia included the upper end of the fibula. The bones in both rabbits show changes that are similar to those in rabbit 317 and its control but are of greater degree. In the long bones two or three neighbouring trabeculae have occasionally been completely removed by osteoclasts in the zone of calcified chondral trabeculae upon which little or no bone has been deposited. In such spaces there is sometimes a delicate fibrous tissue. Elsewhere in this zone the clefts upon either side of the central capillary contain a few osteoclasts but are otherwise filled with osteoblasts. The spongiosa of the metaphysis is embedded in a dense fibrous tissue and contains a remarkable number of osteoclasts in lacunae; it also shows, however, many osteoid zones covered by osteoblasts. In a few bones one or two trabeculae extend from the metaphysis into the medulla. They are clothed partially or completely by a zone of fibrosis. The corticalis for some distance from the epiphysis is greatly reduced and split up by eroded cavities filled with fibrous marrow. In the remainder of the shaft erosion is less, but is shown by conspicuous enlargement of occasional vascular canals within the corticalis and by long shallow cavities upon its inner surface. In general, erosion is greatest near and on the inner surface of the corticalis. Osteoclasts in lacunae are numerous, but many eroded surfaces are covered by osteoblasts upon osteoid zones. The corticalis is bounded within by a zone of fibrosis, except where erosion is absent. The fibrosis is confined to the spongiosa in the metaphysis, to the surface of trabeculae that occasionally extend farther into the medulla, and to the inner surface of the corticalis and to the Haversian spaces eroded within the corticalis. The medullary cavity is otherwise normal, except near the centre of the shaft of the femur of rabbit 318. Here a few small trabeculae of newly-formed bone are embedded in an area of fibrous marrow, and touching the margin of this area is a rounded space which is filled with thin coagulum and has an ill-defined margin. This is the only structure that resembles an early bone-cyst. The bony epiphyses seldom show any abnormality. In some there are a few eroded cavities in the peripheral plate of bone, particularly the part next the junctional cartilage, and a few bays eroded upon trabeculae of the spongiosa. Fibrosis is confined to such eroded cavities and bays. All the long bones appear to be about equally affected. The parietal bones as in rabbit 317 and its control show erosion associated with fibrosis within and on the surfaces of the inner and outer tables, and on the surfaces of trabeculae in the spongiosa. The eroded spaces contain osteoclasts or are lined with osteoblasts upon an osteoid zone.

The activity in growth in length of the long bones appears to be the same in rabbit 318 and its control. As was seen in the macerated specimens, however, the spongiosa forms a very much shallower zone in 318 than in the control. The bones of 318 are in general more porotic than in the control, but they show a less intensely active osteoclastic resorption. Although in the control, therefore, osseous tissue is somewhat more abundant, the bones show the appearances of a more acute and greater osteitis fibrosa.

*Rabbit 319 (770 Units Parathormone), 316 (2150 Units), and Control.*—Time only permitted microscopical examination of two ribs and a longitudinal section of

three vertebrae in each case. The ribs of rabbit 319 show changes that are similar to those in previous animals, but less pronounced. The traeculae in the spongiosa are scanty and short. They are ensheathed by zones of fibrosis which are slightly narrower and less dense than in rabbit 317 and its control. The corticalis throughout the length of the section contains many large spaces that are filled with fibrous marrow and lined with osteoclasts in lacunae. A narrow zone of fibrosis lines most of the inner surface of the corticalis on each side. In rabbit 316 these changes are less marked. The spongiosa appears to be more abundant. There are fewer spaces eroded within the corticalis. The zone of fibrosis upon the inner surface of the corticalis is more focal. In the control rabbit the changes are very similar to those in rabbit 316. There is very little erosion within the corticalis, but there are several large trays, lined by osteoclasts, upon the inner surface. In the bodies of the vertebrae the plates of bone contain a few spaces filled with fibrous marrow and lined with osteoclasts, but otherwise the association of fibrosis with erosion is not seen. In the control rabbit the traeculae in the spongiosa are fewer and thinner than in the injected animals. The corticalis is also thinner, and more interrupted; that forming the posterior wall of the bodies is absent in nearly half of its length.

Judged by the ribs, therefore, the skeleton in this group of rabbits shows changes similar to those in all the others. The changes are of slightly less degree than in rabbit 317, which received 374 units of parathormone, and its control, and of much less degree than in rabbit 318, which received 610 units, and its control. Further, within this group the changes in rabbit 319, which received 770 units, are more pronounced than in rabbit 316, which received 2150 units; and rabbit 316 shows no more change than the control rabbit.

The microscopic examination of the bones of the rabbits in Dr. Byrom's experiments has, therefore, given no unequivocal information concerning the action of parathormone on the skeleton, although the changes seen with the naked-eye appeared to confirm the findings of Bauer, Aub, and Albright. The experiments have, however, shown that in the bones of a whole litter of seven, apparently healthy, young rabbits there were changes identical with those that have been given in detail above as characteristic of generalized osteitis fibrosa in man, except that only one doubtful cyst was observed and there were no osteoclastomata. Analysis gives no evidence that the administration of parathormone influenced the condition in any way. The condition varied with age. It was most pronounced in the animals killed when 19 weeks old, and slightly less at the age of 22 weeks than at 14 weeks. Further work can alone determine the prevalence of the condition, and the influence of diet, etc. The parathyroid glands were not examined. The resemblance of the changes in the skeleton to those in generalized osteitis fibrosa in man is so close that the rabbit may possibly prove an animal in which that disease can conveniently be studied. Lévy (1908) described changes in the skeleton of an adult female rabbit which were essentially similar to those described here. The rabbit had had a portion of caseous mesenteric gland from a child inoculated into the anterior chamber of an eye. It died forty-four days later. The skeleton appeared to be normal to the naked eye, but was examined microscopically because calcareous metastases had been found in internal organs and vessels. The parathyroid glands are said to have shown no microscopical change.

Shortly before Dr. Byrom began these experiments Jaffe and Bodansky (1930) and Jaffe, Bodansky, and Blair (1930) had published in America a preliminary report upon similar experiments upon puppies and guinea-pigs.

According to the details published later in the year these authors found that in young guinea-pigs on ordinary diet a single injection of 20 units of parathormone to 100 grm. of body weight produced in the ribs as early as the twelfth hour osteoclastic lacunar resorption of the corticalis and some fibrosis of the marrow at the costochondral junction. At the eighteenth hour the resorption was greater, the enlarged spaces eroded in the corticalis were filled with fibrous marrow, and formation of new bone at the costochondral junctions had ceased. At the forty-eighth hour there was a spontaneous fracture of the greatly thinned corticalis. In young fasting guinea-pigs similar results were obtained, but in adult guinea-pigs, either fed or starved, there were no changes in the bones that could be attributed to the parathormone. By repeated injections of parathormone over long periods into puppies and young guinea-pigs they produced great general osteoclastic resorption, extensive fibrosis of the marrow, small cysts, and metastatic calcification in the subcutis and viscera. The parathyroid glands were atrophied. In these experiments very little osteoid tissue was found, except at the site of fractures. Experiments were, therefore, devised to allow repair, the injections being discontinued periodically and the animals being killed three to seven days after the last injection. During the period of repair extensive subperiosteal callus was formed, a considerable amount of bone was laid down upon the wide Haversian spaces, and a considerable amount of osteoid tissue was formed at the costochondral junction and in the metaphysis. The osteoid tissue depicted in the shaft of a long tubular bone of a guinea-pig in *Fig. 7* (Jaffe, Bodansky and Blair, 1930) is evidently coarse-fibred.

The authors found necrosis of the cells of the hæmatogenous marrow in all dogs that died from acute hyperparathyroidism. They believed that in acute hyperparathyroidism there was also destruction of other lymphoid tissues. They considered that there could be little doubt that in prolonged treatment with parathormone the fibrosis of the marrow was in part an expression of the healing of injured marrow; they frequently refer to fibrosis of the marrow as scarring of the marrow. But the authors point out as a striking feature the absence of scarring of the marrow in animals that were protected from acute hyperparathyroidism by the administration of preliminary small doses of parathormone. Fibrosis of the marrow appeared at an early stage within the Haversian spaces that were being eroded in the compacta. In such spaces there is no hæmatogenous marrow which by destruction could stimulate fibrosis. Moreover, from their descriptions and illustrations the fibrosis appears to have had exactly the same distribution and intimate relation to bone as in Dr. Byrom's rabbits and in generalized osteitis fibrosa in man. They considered the cysts to be vascular spaces dilated secondarily to disturbances of the circulation.

Jaffe, Bodansky, and Blair, therefore, claim to have caused typical generalized osteitis fibrosa with cysts in young guinea-pigs and dogs by the prolonged administration of parathormone. They thus appear to have proved conclusively the correctness of the interpretation of the changes in the skeleton in man that has been suggested above from examination of the histological processes. Their experiments were very numerous, and evidently performed with great care; in those upon guinea-pigs the bones from nineteen untreated young and old, fed and fasting, animals were studied as controls. Further, the animals were puppies and young guinea-pigs, and not young rabbits. It appears, therefore, unjustifiable to doubt their results, but for anyone who has recently had such a surprising experience as the examination of Dr. Byrom's material unquestioning belief is impossible until the experiments have been confirmed by others. The condition in Dr. Byrom's treated and untreated rabbits appears to have been identical with the condition described by these authors in their guinea-pigs, except that they found little osteoblastic activity when the injections were not interrupted.

It remains to find an explanation of the enlargement of the parathyroid glands. Adding two cases previously published (Hunter, 1930, *Cases 3 and 4*) we have examined six cases. In two cases (*Cases 1 and 4* of this paper) two glands were enlarged. The measurements were: *Case 3*, 1930,  $2 \times 2 \times 2$  cm.; *Case 4*, 1930,  $3.7 \times 3 \times 3$  cm.; *Case 1*,  $2.8 \times 2.5 \times 1.8$  cm. and  $1.1 \times 1.1 \times 1.1$  cm.; *Case 2*,  $2.3 \times 1.5 \times 0.9$  cm. (1.3 gm.); *Case 3*,  $6.8 \times 2.8 \times 1.4$  cm.; (13.5 gm.); *Case 4*,  $7.5 \times 5 \times 1.8$  cm. (26.2 gm.), and  $2 \times 1.3 \times 1.2$  cm. The enlargements are, therefore, in all cases very great and obviously far beyond the normal fluctuations in size. In no case, however, do the enlargements appear to be autonomous new growths. They are occupied by cells which, apart from giant nuclei and multiple nuclei, correspond in structure and size to those found in the normal glands of adults (p. 220). Giant and multiple nuclei are not evidence of neoplasm; they are found in abnormal activities of glands, for instance in the thyroid gland in Graves' disease. In *Case 3*, 1930, and *Case 1*, there is in places a hyaline degeneration of the capsule or of the walls of vessels, which is usually complicated by calcification. In *Case 1* a few parenchymatous cells that are enclosed within the meshes of the net of hyaline calcified vessels are themselves necrosed and calcified. Such retrogressive changes are not, however, proof of neoplasm; degenerative changes are very conspicuous in diffuse colloid goitre. In *Cases 3 and 4*, 1930, and *Case 3* many irregular and rounded cystic spaces are filled with acidophil coagulum. But these spaces are merely exaggerations of the small spherical spaces containing secretion which are frequently found in normal glands. There is no other abnormality in the architecture of the glands except such alterations in the shape of the acini and the disposition of their reticulo-vascular boundaries as would necessarily result from enlargement of the acini by hyperplasia of their cells. The predominant type of cell varies in different cases and in different glands in individual cases, suggesting phases of functional activity. In *Case 3*, 1930, ballooned principal cells greatly predominate, and in *Case 2* they constitute almost all the cells. In *Case 4*, 1930, slightly vacuolated principal cells and pale oxyphil cells are present in about equal numbers. In *Case 3* almost all the cells are pale oxyphil cells with considerable dropsical separation of their granules. In *Case 1* dark oxyphil cells greatly preponderate over ballooned principal cells in the smaller gland, but in the larger gland the relations are reversed so that the ballooned principal cells form the bulk of the two glands taken together. In *Case 4* the larger gland is occupied almost completely by pale oxyphil cells, whilst in the smaller gland small acini of principal cells are clearly in process of conversion into large acini of pale oxyphil cells. The ballooned principal cells contain, as in the normal gland, much glycogen. The pale oxyphil cells contain less glycogen, the dropsical type (*Case 3*, *Fig. 190*) containing more than the more solid type. The evidence that the enlargement is a functional hyperplasia appears to be conclusive in *Case 4*, where the smaller gland shows all stages of conversion of small acini of principal cells into massive acini of pale oxyphil cells similar to the acini that occupy the larger gland.

The evidence appears to be conclusive that the enlargement of the parathyroid glands is due to a functional hyperactivity and not to autonomous



neoplasm. Consequently, if the changes in the bones are the result of excessive secretion of the parathyroid glands, the position is similar to that in Graves' disease, in which the cause of the abnormal activity of the thyroid gland is unknown. Parathyroid hyperplasia is known, however, to occur secondarily to abnormal conditions of the skeleton. It has been seen, for instance, in the disturbance of calcium metabolism constituting osteomalacia (Erdheim, 1907; Hoffheinz, 1925), and in hæmatogenous myelomatosis (Barr and Bulger, 1930; Bulger, Dixon, and Barr, 1930). Further, Klemperer (1923) found hyperplasia of a parathyroid gland in a case of extensive destruction of the skeleton by secondary carcinoma, but he thought that osteomalacia was also present. The view that the enlargement of the parathyroid glands in generalized osteitis fibrosa is secondary to the changes in the bones cannot, therefore, be dismissed without consideration. In focal osteitis fibrosa there is no generalized osteoclastic resorption of the skeleton. The changes, however, in the bone in the areas affected by the focal osteitis fibrosa cannot be distinguished histologically from those in the more severely affected areas in generalized osteitis fibrosa, as is shown by *Case 8* of this paper. Cysts and osteoclastomata frequently occur. In Paget's osteitis deformans the resemblance to generalized osteitis fibrosa is still greater because the alteration may involve the whole skeleton. Von Recklinghausen (1891), who first described under the name of "fibrous or deforming ostitis" three cases, of which one at any rate (*Case VII*) was a typical example of generalized osteitis fibrosa, considered that his cases were a form of Paget's osteitis deformans. The resemblance is recognized by the two conditions being called respectively osteitis deformans of Paget and osteitis deformans of von Recklinghausen.

In Paget's osteitis deformans there is a similar interplay of excessive lacunar resorption and apposition. Even in cases in which the disease has been conspicuous for years osteoclasts in Howship's lacunæ may be very numerous in parts of the bones. In the descriptions in this paper of the portions of bone removed in generalized osteitis fibrosa stages of reconstruction of the compacta can be traced: porosis is initiated by lacunar resorption of the Haversian canals, whilst continued resorption and apposition lead to conversion of the compacta into a spongiosa that has an entirely new architecture and is composed entirely of bone formed during the disease. In the same way in Paget's osteitis deformans the corticalis of the large tubular bones is converted into a spongiosa of newly formed trabeculae composed of numerous systems of lamellar bone, and the flat bones such as the sternum and calvaria become spongy throughout. Resorption and apposition also reconstruct the trabeculae of the spongiosa, but alterations in the architecture of the spongiosa of the long bones are much less conspicuous than in the corticalis. The newly formed bone in generalized osteitis fibrosa consists largely of woven bone. In the more chronic reconstruction in Paget's disease woven bone is very rare. It is found, however, in places in the earlier stages, but is soon replaced by lamellar bone (Schmorl, 1930). There is no evidence of osteomalacia. The resorption and apposition are associated with a fibrosis of the marrow, and this shows the same intimate relation to bone and to the degree of alteration of bony structure that has been described above as characteristic of generalized osteitis fibrosa. This relation is most

conspicuous in the spongiosa of the large tubular bones, where most of the trabeculae are clothed partially or completely by a zone of fibrous marrow of variable depth; the fibrosis is sometimes confined to a space eroded within a trabecula. The zonal arrangement of the fibrosis can also be recognized in the altered compacta. Engorgement, haemorrhage, and the accumulation of pigment-granule-cells are again conspicuous. Osteoclastomata are rare; Schmorl (1930) has found none. They do occur occasionally, however. In a typical skeleton (P.M. 944, 1907) from a man of 49, who had noticed the deformities for eight years, an osteoclastoma in the skull had extended into the scalp and had also bulged into the brain. Cysts are also rare. Knaggs (1926) found no evidence in museum specimens, and Schmorl (1930) has not seen them in his abundant material. In the London Hospital Museum, however, there is a left femur in which two superimposed cysts, measuring  $2.2 \times 1.4$  cm. and  $2.1 \times 1.4$  cm., are separated by a narrow partition and lie in the inner part of the shaft immediately below the neck. Their inner ends lie in concavities in the corticalis. This specimen was obtained at necropsy (P.M. 363, 1928) from a typical skeleton in a man of 61 years. Spontaneous fractures are relatively common. There is, therefore, a close resemblance between the histological processes in the osteitis deformans of Paget and in generalized osteitis fibrosa. In generalized osteitis fibrosa, however, resorption greatly exceeds apposition, whilst in Paget's osteitis deformans the two processes are more evenly balanced and apposition exceeds resorption. Further, the processes are less acute in Paget's disease. The long continuation of the two processes side by side in very nearly equal amounts is shown by the bone consisting of an abnormally rich mosaic of very numerous small lamellar systems with eroded margins. To this feature, which I have pointed out in my lectures for many years, my former teacher Professor Schmorl (1930) has drawn particular attention; he considers the great number and the irregular arrangement of the systems to be diagnostic of the disease. In the description in this paper of the portions of bone excised from cases of generalized osteitis fibrosa the number of systems in the bone formed during the disease has been mentioned, and has been used as a measure of the persistence of both processes of resorption and apposition; but where the systems were most numerous their number was small. The preponderance of apposition is shown in Paget's disease by the characteristic thickening and enlargement of the affected bones and the increase in their weight in spite of the porosity of the compacta. In generalized osteitis fibrosa certain bones, for instance the calvaria in *Case 1*, may be greatly thickened and increased in weight, but the bony skeleton is greatly reduced as a whole. In focal osteitis fibrosa, even when the foci are multiple, and in Paget's osteitis deformans there is no chemical evidence of a disturbance of calcium metabolism (Hunter, 1930). The parathyroid glands are not enlarged in Paget's disease (Schmorl, 1930).

It has been shown that the enlargement of the parathyroid glands in generalized osteitis fibrosa is due to functional hyperplasia, that hyperplasia of the parathyroid glands is known to occur secondarily in diseases of bone, and that the abnormal processes in the bones in generalized osteitis fibrosa and Paget's osteitis deformans are closely similar except that resorption

greatly preponderates in the former. Even if the animal experiments of Jaffe and his collaborators are not accepted as conclusive, the abnormal calcium metabolism in patients, the effect of extirpation of their enlarged glands, and the mobilization by parathormone of metals from the skeleton into the blood make it impossible to believe that the secretion from the enlarged parathyroid glands in generalized osteitis fibrosa does not cause a change in the skeleton. In as much as there is no unequivocal evidence that halisteresis occurs in any abnormality of the skeleton, there can be little doubt that this change is a lacunar resorption. It might, therefore, be argued that generalized osteitis fibrosa and Paget's osteitis deformans both begin with one and the same disease of bone, but that in generalized osteitis fibrosa this disease happens to induce secondarily a functional hyperplasia of the parathyroid glands and the additional lacunar resorption due to parathormone causes the difference between the two conditions. But the cause of the changes in Paget's disease is unknown. Arguments have been given above to show that if the cause of the changes in the skeleton in generalized osteitis fibrosa lies within the bone, it must lie within bone. These arguments apply equally to Paget's disease. It has, indeed, been suggested that Paget's disease is due to some abnormality in the bony substance. By no histological method, however, has any abnormality been demonstrated (Schmorl, 1930). It would be more reasonable to argue from the known to the unknown. There is definite evidence that parathormone alters the normal equilibrium of the skeleton, and that extirpation of enlarged parathyroid glands improves the condition of the skeleton in patients suffering from generalized osteitis fibrosa. All the available evidence points to excessive parathyroid secretion as the cause of the skeletal changes in this disease. The general resemblance to the abnormal processes in Paget's osteitis deformans suggests that the cause of this disease also lies outside the bone, and is some disturbance of the multiple factors which regulate the formation of bone. If it is accepted, however, that the changes in the skeleton in generalized osteitis fibrosa are entirely secondary to hyperfunction of the parathyroid glands, the cause of that hyperfunction is still unexplained. The position, as already mentioned, is similar to that in Graves' disease. The practical importance of this conclusion is very great, because it makes it impossible to foretell the ultimate results of extirpation of one or two enlarged glands.—H. M. T.

### FOCAL OSTEITIS FIBROSA.

Of much more common occurrence than the generalized disease is focal osteitis fibrosa. This is a condition affecting one or more bones, usually not disabling, of slow progress, and showing a tendency to become arrested. It occurs chiefly in adolescence, and is often symptomless until spontaneous fracture occurs. In the descriptions of cases which follow it will be seen that the figures for serum calcium and plasma phosphorus are invariably normal, a finding in striking contrast to that of the generalized disease. The calcium balance is usually normal, and, taken in conjunction with a normal blood chemistry, this finding is of far-reaching importance. Supported as it is by the normal density of the radiographic shadows of bones, it is evidence

strongly against hyperparathyroidism. It is of vital importance in such cases to realize that, although many bones are affected, the condition is totally different from the generalized disease, and that exploration of the neck is usually unjustifiable.

*Case 5.—Osteitis fibrosa in multiple foci.*

O. U., female, aged 18. (L.H. Reg. No. 41679/1928.)

**HISTORY.**—Two years, aching pain in right leg below knee, noticed thickening of right shin. Eighteen months, pain and limitation of movement in right hip. Spontaneous fracture of right femur. After rest in bed for three months good union occurred. Six months, increase in size of swelling on shin with pain. No family history of bone disease.

**ON EXAMINATION.**—Well-developed girl. Calvaria normal. Sclerotics white. No exophthalmos. Lenses normal on examination by slit-lamp. Hearing normal. Teeth sound. Slight uniform (adolescent) thyroid enlargement. No other swelling felt in neck. No tachycardia. No limitation of movement of hips or other joints. Slight thickening but no bowing of subcutaneous surface of right tibia in its middle third. Urine normal. Wassermann reaction negative.

**Radiograms.**—Pale cyst-like areas in upper two-thirds of shaft of right femur and in middle third of shaft of tibia, the latter expanding the corticalis. Controlled radiograms showed that outside these areas the shadows of all bones examined were of normal density. Calvaria normal. No calculi in renal tract (Dr. S. G. Scott).

*The blood chemistry* was examined at intervals over a period of two years and found repeatedly to be normal (serum Ca 9.9 mgrm. per 100 c.c., plasma P 3.5 mgrm., plasma phosphatase, 0.248 mgrm.). On a known low calcium intake the calcium output in urine and faeces was normal (*Fig. 180*).

*Case 6.—Focal osteitis fibrosa of right humerus.*

R. N., male, aged 13. (L.H. Reg. No. 31663/1930.)

**HISTORY.**—Two years ago while running he slipped and fell backwards hurting his right shoulder. Humerus immobilized for four days in plaster jacket. Treated subsequently by massage. Three months, occasional sharp pain at point of right shoulder, unrelated to movement and with no radiation. No pain elsewhere. No family history of bone disease.

**ON EXAMINATION.**—Thin, rather small boy. Calvaria normal size and contour. Sclerotics white. Lenses normal on examination by slit-lamp. Hearing normal. Teeth good. No tumour felt in neck. No abnormality of skeleton detected. Urine normal. Wassermann reaction negative.

**Radiograms.**—Pale area with multiple trabeculae expanding corticalis in upper third of right humerus. Controlled radiograms showed that outside these areas the shadows of all bones examined were of normal density. Calvaria normal. The renal tract showed no calculi.

*The blood chemistry* was found repeatedly to be normal. (Serum Ca 10.0 mgrm. per 100 c.c., plasma P 3.8 mgrm., plasma phosphatase 0.379 mgrm.) On a known low calcium intake the calcium output in urine and faeces was normal (*Fig. 180*).

*Case 7.—Osteitis fibrosa in multiple foci. (Hunter, 1930, Case 5.)*

L. R., married woman, aged 27. (L.H. Reg. No. 40007/1930.)

**HISTORY.**—Twelve months, enlargement of right arm above elbow. Three weeks, aching pain in arm began in fourth month of pregnancy. No fracture or history of fracture. No family history of bone disease. Two children well.

**ON EXAMINATION.**—Well-nourished, small woman. Calvaria normal. Sclerotics white. No visual defect. Slit-lamp examination showed within the foetal nucleus of each lens crowds of tiny white glistening dots surrounded by a small area of cloudy lens (Mr. C. Goulden). Hearing normal. Caries of three teeth. No tumour

felt in neck. Diffuse irregular enlargement of right humerus with outward bowing in upper part. No deformity of other bones. Urine normal. Wassermann reaction negative.

*Radiograms.*—Right humerus and radius show pale trabeculated areas with expansion of bone. Similar, less definite, areas round acetabulum and in left ischium. Controlled radiograms showed that outside these areas the shadows of all bones examined were of normal density. Calvaria normal. The renal tract showed no calculi (Dr. S. G. Scott).

*The blood chemistry* was found repeatedly to be normal (serum Ca 9.8 mgrm. per 100 c.c., plasma P 3.0 mgrm., phosphatase 0.363 mgrm.). On a known low calcium intake the calcium output in urine and faeces was normal (*Fig. 190*).

#### *Case 8.—Osteitis fibrosa in multiple foci.*

G. B., male, aged 12. (L.H. Reg. No. 31553/1930.)

*HISTORY.*—Two years, pushed on to curb-stone, broke left arm. Treated for fracture of left radius by splinting and massage. Twelve months, mother noticed that he limped with the right leg and she found right thigh bowed outwards.

*PAST HISTORY.*—Lump on back of head for years. Never had rickets. No history of polydipsia, polyuria, renal pain, or passage of stone or gravel. No family history suggesting disease of bones.

*ON EXAMINATION.*—Well-built boy. Abnormal prominence of left side of occipital bone and of left parietal eminence. Sclerotics white. Lenses normal on examination by slit-lamp. Hearing normal. Teeth good. No tumour felt in neck. Right femur bowed outwards and forwards; right leg 1.5 cm. shorter than left. Thickening of second phalanges of third and fourth digits of left hand. No tenderness of bones on pressure. Heart, lungs, abdomen, and nervous system normal. Trousseau and Chvostek signs absent. Urine normal. Wassermann reaction negative.

*Radiograms.*—Shadow in left parieto-occipital region suggests osteoma. Bowing of right femur with new bone formation along inner aspect and pathological fracture through thinned, irregularly mottled outer aspect. Left radius, left humerus and metacarpals, and most phalanges of left hand show pale irregular shadows with slight expansion of the corticalis suggesting osteitis fibrosa. Controlled radiograms show that outside these areas the shadows of all bones examined are of normal density. No renal calculi (Dr. G. E. Vilvandré).

*OPERATION* (Mr. R. Milne—July 14, 1930).—Wedge-shaped osteotomy of right femur performed through middle of antero-lateral aspect of thigh. Alignment of limb corrected; right hip immobilized in plaster-of-Paris.

### **Pathological Report.**

(PROFESSOR H. M. TURNBULL.)

Tissue removed at operation. (S.D. 1626/1930.)

#### **1. Right Femur.—**

*MACROSCOPIC.*—The specimen was a wedge-shaped segment (3 × 2 × 2 cm.) of the whole cross-section of the shaft. It had been placed in formaldehyde. The cortical surfaces, where not covered by periosteum, were grey and beset with longitudinal sulci that were just visible to the naked eye. On the cut surfaces the inner and outer corticalis formed zones (up to 2 mm. deep) of homogeneous grey bone interrupted by a few small pink dots and streaks. Between these was a dense, gritty, cancellous bone in which a net of slender grey trabeculae, enclosing small pink spaces, was just visible to the naked eye. The spaces tended to have their long axes parallel to the cortical surfaces.

*MICROSCOPIC.*—The wedge was fixed and extensively decalcified by alternate immersions in 4 per cent formaldehyde in distilled water (pH 3 to 4) and Müller's fluid, followed finally by thirty minutes in 5 per cent nitric acid.

A narrow layer of corticalis occupies both the base and the apex of the wedge. It has a spongy texture owing to large medullary spaces occupied by fibrotic marrow. On one side a portion of tendon replaces the periosteum and beneath this are areas of woven coarse-fibred bone and, rarely, of calcified fibrocartilage. The rest of this cortical layer consists of lamellar bone. Recent resorption is shown by many large osteoclasts in Howship's lacunæ upon the walls of some of the medullary spaces. Recent apposition is shown in several places by osteoblasts upon osteoid zones. Extending from one corticalis to the other is a close mesh of slender bony trabeculæ in a fibrotic marrow (*Fig. 188*). The trabeculæ are composed of woven bone, but near the corticalis a few contain small portions of lamellar bone. Many trabeculæ are formed by more than one system of woven bone. Recent resorption is shown throughout by many large osteoclasts in lacunæ, and past resorption by numerous lacunæ beneath osteoid zones. Apposition is, however, at present greater than resorption. It is very active, the osteoblasts being very numerous and large. The osteoid zones in general appear to be very deep (*Fig. 188*). This is due largely to frequent obliquity of section. Where cut at right angles to their surfaces the zones are not deeper than in actively healing fractures. The marrow in the medullary spaces of the corticalis and medulla consists of numerous spindle and stellate fibroblasts in a matrix of closely packed collagen fibrils. The fibroblasts are very uniform in morphology; variations in the density of the fibrosis are slight; the vessels have walls of normal structure and are not dilated.

**Remarks.**—Resorption has led to the disappearance of almost all the original bone except a narrow outer strip of corticalis, and has converted this from compact bone into spongy. In the rest of the bone resorption and apposition have replaced lamellar bone by a woven bone, and have completely altered the architecture. The new spongiosa is of much closer texture than the normal and the medullary cavity has been obliterated. Long duration of abnormal conditions is shown by many trabeculæ containing a mosaic of separate systems of woven bone. These changes are associated with a dense but cellular fibrosis of the marrow. There is no evidence of a cessation of calcification of osteoid tissue, that is, of osteomalacia. The general picture is that of an osteitis fibrosa. The changes cannot be differentiated from those in the osteosclerotic areas in generalized osteitis fibrosa—for instance, in the skull in *Case 1* (*cf. Figs. 188 and 187*).—H. M. T.

**SUBSEQUENT PROGRESS.**—The patient was sent to a convalescent home and later re-admitted for further investigation.

**Calcium and Phosphorus Metabolism.**—The serum calcium was constant within very narrow limits, the average figure for eight estimations being 10.4 mgrm. per 100 c.c. The corresponding figure for plasma phosphorus was 3.5 mgrm. Nevertheless the plasma phosphatase figure was always high, the average for six estimations being 0.912 mgrm. The calcium output was estimated in the urine and feces for three three-day periods. The patient was kept on a weighed diet of known low calcium content. The calcium output in the urine was about twice the normal, that in the feces being also about twice the normal (*Fig. 180*).

**OPERATION** (Mr. E. C. Lindsay—Oct. 22, 1930).—Under open anæsthesia collar incision made in neck. Sternomastoid muscles were retracted on either side and pretracheal muscles divided. Superficial lymphatic gland removed for section. Thyroid gland apparently normal. Three normal parathyroid bodies identified, left superior not seen. No parathyroid tumour discovered after extensive search. Wound closed. A portion of bone was removed from the lower end of the left radius.

## Pathological Report.

(PROFESSOR H. M. TURNBULL.)

Tissues removed at operation. (S.D. 2428/1930.)

## 1. Left Radius.—

**MACROSCOPIC.**—A longitudinal slice 2.6 cm. long and 0.6 cm. deep in the centre. The outer surface was covered with periosteum. The cut surface showed a hard white sheet of corticalis, too shallow to be measured, and beneath this a deep red, gritty, and granular tissue in which a spongy texture was just recognizable.

**MICROSCOPIC.**—The specimen was decalcified completely almost throughout by Müller's fluid followed by nitric acid.

The corticalis has been reduced to a shallow shell, which is split in most of its extent into two layers by a row of long medullary spaces directed in the long axis of the bone. It consists of lamellar bone (*Fig. 197*). Beneath it is a spongiosa formed entirely of woven bone (*Fig. 198*) which is usually coarse-fibred in the centre of the trabeculae and of intermediate type in the periphery. The trabeculae are more slender and form a slightly closer net than in the bone removed at the first operation. Further, they show very few separate systems. Otherwise the appearances throughout are very similar. As before, there is much active resorption by osteoclasts, but apposition greatly preponderates. The edges of the bone beneath the osteoid zones are beset with lacunae. The osteoid zones taken as a whole are very conspicuous, but where they are cut at right angles to their surface the depth is not excessive in view of the activity and the type of bone-formation. The marrow shows a similar fibrosis.

**Remarks.**—The changes are similar to those in the first piece of bone, but the abnormal changes appear to have been present for a shorter period, the trabeculae of woven bone showing very few separate systems. The changes are such as are found in osteosclerotic areas in examples of generalized osteitis fibrosa.

**2. Lymphatic Gland.**—The gland measured  $1.3 \times 0.7 \times 0.4$  cm., and its lymphatic nature was confirmed by microscopic examination.—H. M. T.

**Remarks.**—The points against hyperparathyroidism in this case are the normal serum calcium and plasma phosphorus together with the absence of evidence of osteoporosis in controlled radiograms. The high calcium output and high plasma phosphatase are probably expressions of the destruction of bone in multiple foci. Future estimations of calcium metabolism in this case will afford information of great value. It is not known whether in such cases hyperparathyroidism ever develops.

## OSTEOMALACIA.

Morbid anatomists are agreed that in rickets and osteomalacia the essential abnormality is the same—namely, a deficient calcification of osteoid tissue. This deficiency is generalized throughout the skeleton. In rickets, since endochondral ossification has not ceased, the zone of provisional calcification is affected too, and endochondral ossification is irregular.

The nature of the calcium deficiency has given rise to much controversy. Virchow (1853) held the view that calcium salts had been removed from calcified bone, leaving osteoid tissue. This was the doctrine of 'halisteresis',

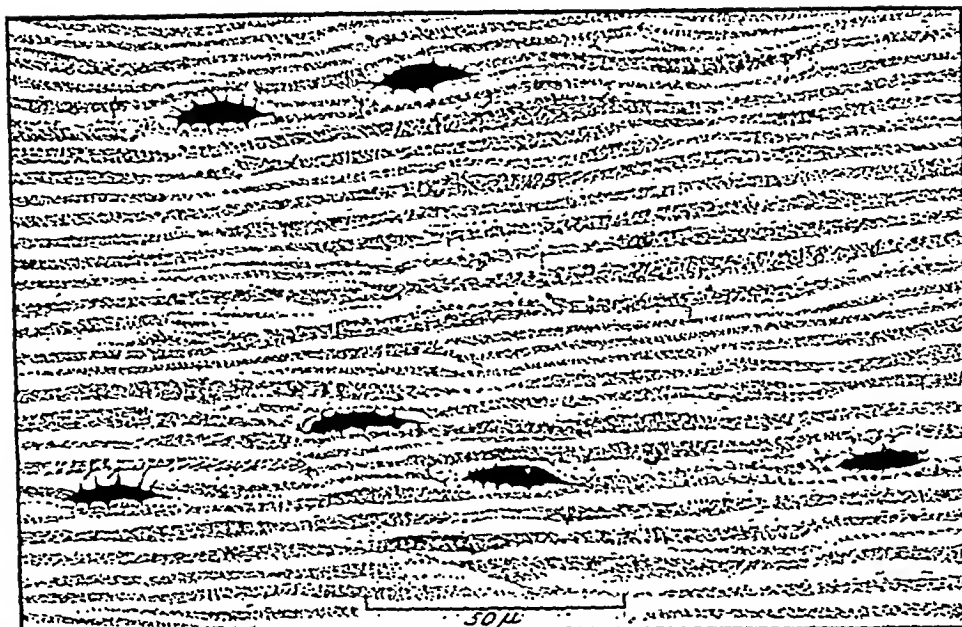


FIG. 197.—Case 8. Left radius. Osteitis fibrosa in multiple foci. Müller's fluid followed by nitric acid; Schmorl's thionin-phosphotungstic-acid method for fibrils. Lamellar bone in outer remnant of compacta of corticalis.

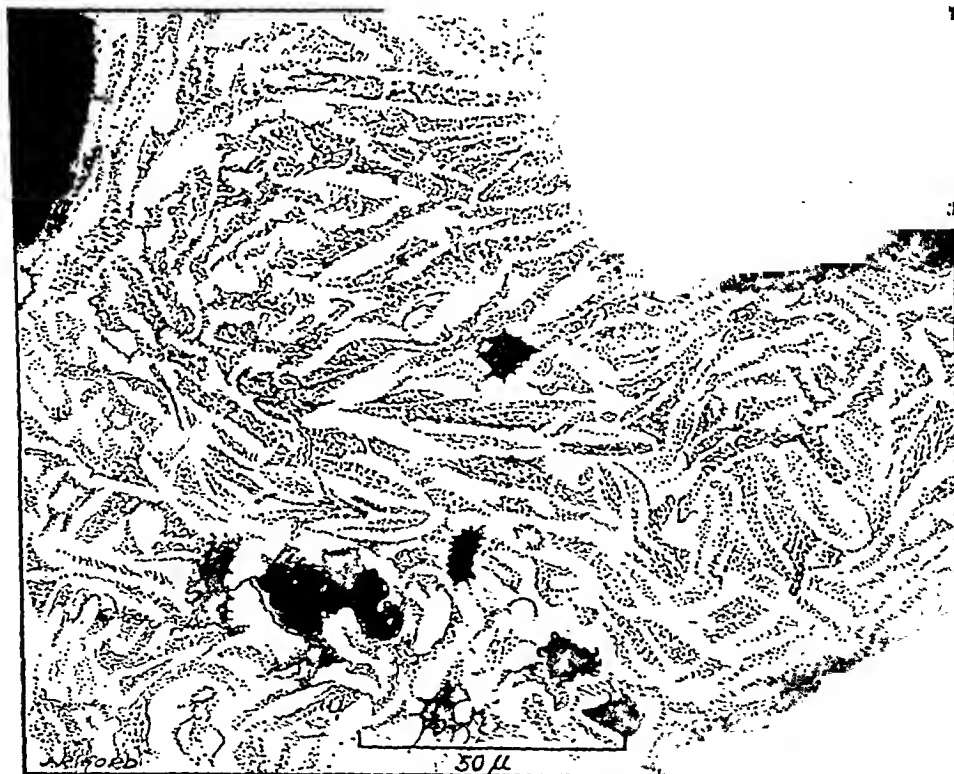


FIG. 198.—Case 8. Left radius. Osteitis fibrosa in multiple foci. Müller's fluid followed by nitric acid; Schmorl's thionin-phosphotungstic-acid method for fibrils. Trabecula of woven, coarse-fibred bone in newly formed, spongy corticalis beneath.



which means literally 'salt deprivation'. Pommer (1885) and Schmorl (1909), while expressing the view that osteomalacia and rickets are essentially the same disease, held in addition that haliteresis is not responsible for the broad osteoid zones. They considered that, in both diseases, the calcifying mechanism which should convert osteoid tissue into true bone was deficient. Not only does any bone added by advancing endochondral ossification remain osteoid, but so also does all new bone added to trabeculae which were fully calcified at the onset of the disease. The normal physiological resorption accompanies this apposition, and consequently portions previously hard become ever softer and more liable to bend.

Now, although there are many problems still unsolved concerning the etiology of these two diseases, it is clear that the calcifying substance which is missing is vitamin D. In its absence the utilization of calcium and phosphorus is impaired. In the majority of cases of osteomalacia the serum calcium is low as in low-calcium rickets. In both diseases the abnormalities in the blood are promptly restored to normal by the use of cod-liver oil, ultra-violet light, or irradiated ergosterol. At the same time ossification begins to proceed normally. Biochemistry has therefore amply justified the view held by Pommer and Schmorl of the pathogenesis of these diseases and their essential identity.

**Clinical Features.**—Osteomalacia is a deficiency disease endemic over wide areas in Northern India, Japan, and Northern China, and occurring sporadically in the Rhine Valley, Danube Valley, Vienna, and certain parts of Italy, Switzerland, Flanders, and the Balkans. It is a rare disease in the British Isles and U.S.A. It pre-eminently affects women, and is likely to recur earlier and with greater severity in each successive pregnancy. Many cases clear up after lactation is complete, or even before this time. However, it is a mistake to suppose that pregnancy is essential in its etiology; it is sometimes seen at puberty, and is quite well known to occur, though it is rare, in boys and men (Wilson, 1931). Maxwell (1923) states that it is known to the Chinese as 'Yao t'ui t'eng'—'back-and-thigh pain'. The pain is aching in character and is worse in the winter months. The pelvis, thorax, or long bones show deformity in a haphazard way; one woman suffers in the pelvis, another in the ribs, and a third in both. Besides the characteristic and well-known change in the pelvis, marked deformities occur in the chest and spine. Severe kypho-scoliosis may reduce the height by several inches and cause the head and neck to sink downwards and forwards on to the chest. Deformities of the sternum and ribs may give rise to marked prominences and depressions in the chest wall. Coxa vara and irregular curves in the long bones are less common. The bones are soft and flexible rather than fragile, so that bending is much more common than spontaneous fracture, though both are well recognized. The patient develops a characteristic waddling gait, and muscular weakness may add to her incapacity. In many cases the pelvic deformities interfere with marital relations or with labour, Cæsarean section frequently being necessary. Tetany is very common, and the attacks may last for hours. Wilson (1931) has found evidence of osteomalacia in cases of the so-called idiopathic tetany or 'wai' of Kashmir.

**Pathogenesis.**—It is only in recent years that real progress has been made towards solving the problem of the etiology of this condition. For almost half a century the hypothesis was held that the disease was due to a pathological increase in the activity of the ovaries leading to hyperæmia of the bones and a consequent solution of their lime salts. This was not based on accurate experiment, neither was there any evidence worth the name that the ovaries were histologically abnormal. The hypothesis arose in 1879 when Foehier performed Cæsarean hysterectomy in several cases of osteomalacia and found that a good effect was produced on the progress of the disease. Fehling (1884) performed ovariectomy for osteomalacia quite apart from pregnancy, and within a few years this treatment was performed all over the world. Now, while there can be no doubt that pregnancy leads to the development of osteomalacia, there is very little evidence that the ovaries exert any effect on the disease apart from their rôle in pregnancy. Castration presumably acts by stopping the possibility of further pregnancy and lactation with their consequent drain on skeletal calcium, and this can be as easily done by ligature of the Fallopian tubes.

An amazing fallacy regarding the etiology of osteomalacia originated in the work of Bossi (1907), who arrived at a method for treating the disease with adrenalin by the following argument: "Since suprarenal substance is known through its pressor action to modify deeply the circulation in both sexual organs and bone-marrow, since removal of the suprarenal has been shown in animals to cause deep and lasting changes in the ovary, I thought of treating this case with suprarenal substance (adrenalin)." After claiming, on quite inadequate evidence, that the patient was cured by the injections, he proceeded to an experiment upon a sheep in the middle of pregnancy. He removed one suprarenal and the animal remained well until the seventh day, when it had difficulty in walking. On the eighth day it could neither stand nor walk, and it showed "curvatures of the joints in the whole body, the hips markedly rotated inwards, and painful on pressure". Such is the evidence on which the suprarenal origin of osteomalacia is based. One asks oneself whether its exponents of to-day have troubled to read the article from which it sprang!

Studies of the mineral metabolism of osteomalacia date back many years, and began with analyses of the bones. McCrudden (1910) in a case of adolescent osteomalacia found 15.4 per cent CaO in dried bone as compared to 28.8 per cent in normal bone. In 1910 the same observer demonstrated a negative calcium balance in eight cases of osteomalacia.

The blood chemistry had not been studied accurately until Miles and Feng (1925) investigated ten patients. These cases varied in duration from eight months to twenty years; in seven there was tetany, and in all a considerable diminution in the serum calcium (5.0 to 7.4 mgrm. per 100 c.c.). The plasma phosphorus varied from 1.8 to 3.8 mgrm. per 100 c.c. The same workers carried out calcium balance experiments on four adult females taking a low calcium diet consisting of millet gruel, wheat bread rolls, celery-cabbage, garlic, and dry steamed millet. A negative calcium balance was found in three patients, positive in one. Keeping the diet identical and adding cod-liver oil, alone or in combination with either calcium lactate or calcium

phosphate, they were able to bring about marked retention. In one patient receiving olive oil with calcium lactate, the calcium balance remained negative. Osteomalacia therefore chemically resembles low calcium rickets. In the present state of our knowledge the occurrence of hyperplasia of the parathyroids in osteomalacia (Erdheim, 1907) is without explanation. Since all the parathyroids are usually affected, the hypothesis that the change is compensatory seems attractive.

**Economic Factors Responsible for Osteomalacia.**—With the story of rickets before us we can trace the economic factors operating in the causation of osteomalacia. They are factors difficult for a Western civilization to appreciate, deep-rooted as they are in the social and religious customs of the East. The diet of the Chinese people is meagre, consisting, as in the experimental diet used by Miles and Feng, entirely of cereals and a limited amount of vegetables with no milk, meat, or eggs. It is deficient in vitamin D and in calcium salts, and contains an excess of acid-forming over base-forming mineral elements. The environment tends to keep the woman patient isolated from the sun, for foot-binding retards movement, and the severe winters of the high plateau of North China lead to confinement indoors. When the pains of osteomalacia begin she retires to her warm brick bed, thereby completely shutting herself off from the sun. In India the areas affected are in the north, in the high valleys where sunlight is scanty in winter owing to the surrounding mountains. As in China, there is a marked seasonal incidence, the disease being worse in winter and early spring. In Kashmir the disease is worse in the cities. There are narrow courtyards and the houses have a minimum of window space. The women affected are those observing strict purdah (Vaughan, 1926). They are systematically screened from the sun from the age of ten. No cases are seen in Kashmiri boat women who live in the open air and drink milk provided by a goat or a cow carried in the boat. Nevertheless, Wilson (1931) has observed in the Kangra district of the Punjab severe osteomalacia in women who work in the open air where sunshine is plentiful for eight months of the year. The diet of these women is grossly inadequate and contains no milk or eggs. In many places in India no raw fruit or vegetables are eaten, largely from fear of cholera. The pregnant woman is kept short of food with the idea of keeping the foetus small for an easy labour. The calcium drain of lactation is very severe, for it commonly continues as long as four years. Under such social conditions it is not surprising that other deficiency diseases may exist together with osteomalacia. Thus Wampler (1924) reports oedema in one-third of forty-six cases, and xerophthalmia in one.

**Treatment.**—In the treatment of osteomalacia, as in the case of rickets, cod-liver oil, sunlight, and irradiated ergosterol each has its place. Vaughan reports that "out-patients given cod-liver oil and ordered to go out on the lake in open boats improved rapidly." Starlinger (1927) and Hottinger (1927) report cures with irradiated ergosterol, and it is an interesting fact that each of their cases had been previously treated unsuccessfully by ovariectomy. Maxwell states that "given cod-liver oil, good food, and sunlight, the majority of the patients will slowly recover. Cod-liver oil and calcium salts quickly remove tetany and the worst of the pain." He finds that some

cases require  $1\frac{1}{2}$  oz. of cod-liver oil with 1 mgrm. of irradiated ergosterol daily. But he adds, "we want flocks and herds, milk and meat, with security of life and property."

*Case 9.—Osteomalacia.*

E. R., married woman, aged 33. (L.H. Reg. No. 40829/1930.)

OBSTETRIC HISTORY.—Catamenia commenced at 13 and were regular. Married 15 years.

1916.—Full term pregnancy, normal labour. Girl, now 14.

1918.—Full term pregnancy, normal labour. Girl, now 12.

1919.—Full term pregnancy, normal labour. Male child; died in convulsions a few days after birth.

1922.—Full term twin pregnancy, normal labour. Boy, now 7. Girl died 17 days after birth, in convulsions.

1923.—Full term twin pregnancy, difficult labour, instrumental delivery. Intra-natal death of girl. Boy survived three months, then wasted and died in convulsions.

The three babies who survived were breast fed for twelve months.

HISTORY.—1922.—Immediately following fourth pregnancy began to have pains in left hip and back. Limped with left leg. Pains in ankles.

1923.—During last month of fifth pregnancy pains in limbs recurred. Vomiting. Was in bed three months with swelling of left leg. Aching all over, especially in lower part of spine. When allowed up could only get from chair to chair with difficulty.

1925.—She was thought to suffer from tuberculosis of the spine, but when radiograms were taken she was told that the condition was osteomalacia, the 'pelvic diameter' being  $1\frac{1}{2}$  in. In bed six months in order to straighten out the bones. Pain in bones on shaking hands.

1926.—Treated with ultra-violet rays and thyroid tablets. Took the tablets twice a day for twelve months, and then her husband made her give them up because her bones were worse; often in bed with pains in bones. From this time husband had to look after house and children.

1928.—Diminution of two inches in total height. Arms and legs tender on deep pressure. Bowing of right forearm. Unable to walk unaided. Able to get about by resting arms on a wheel chair. Five teeth extracted for apical abscesses.

Six months.—Slight bowing of left forearm. Severe aching pains in bones. Muscular weakness, especially in back.

*Past History.*—Born in Darlington, Durham, and has always lived there. Did not suffer from rickets as a baby. Good teeth as a child, first extractions at fourteen for apical abscesses. Has always taken normal food; no idiosyncrasies in regard to diet. No history of having passed gravel or stone. No increased frequency of micturition. No history of fractures nor tetany. No family history suggesting disease of bones.

ON EXAMINATION.—Height, 4 ft. 4 in. Weight, 7 st. 1 lb. Well-nourished woman. Acne rosacea. Able to stand unaided, but requires assistance to walk. Calvaria normal size and contour. Sclerotics white. Lenses normal on examination by slit-lamp. Hearing normal. Double dentures, recession of gums of remaining lower teeth. Tongue clean. Mucous membranes normal colour. No tumour felt in neck. Heart and lungs normal. Blood-pressure 115/85 mm. mercury. No abnormality in nervous system. Trousseau and Chvostek signs absent. Stooping attitude with slight kyphosis and sinking of chin towards chest. Angulation forwards and central thickening of both clavicles. Considerable bowing of right forearm. Thorax sunken towards pelvis; transverse crease in abdominal wall. Genu valgum; when the internal condyles of the femora are approximated the internal malleoli are separated by a distance of  $4\frac{1}{2}$  in. Tenderness on pressure upon long bones and ribs. Points of maximum tenderness, for example, in lower end of right radius and upper end of left fibula. No bone tumours felt. Pelvic measurements: interspinous, 25.4 cm., intercrystal, 28 cm., external conjugate, 16.5 cm., transverse diameter at outlet, 2.5 cm., promontory not reached by examining finger.

*Urine.*—Acid ; sp. gr., 1022 ; no albumin ; no sugar. Bence Jones protein absent.

*Blood.*—Red cells, 5,400,000. Hb, 85 per cent ; C.I., 0.78 ; leucocytes, 7000 ; differential count, normal. Wassermann reaction negative.

*Fractional Test-meal.*—Complete achlorhydria. Total acidity, 10. Slow rate of emptying.

Fat in faeces : total, 37.4 per cent ; unsoaped, 12.7 per cent.



FIG. 199.—Case 9. Osteomalacia. Controlled radiograph of left forearm.

*Radiograms of Bones* (June 5, 1930).—Spontaneous fractures, with no displacement, in shafts of right femur, right fibula, left radius (Fig. 199), in both ulnae, and in 6th, 7th, 9th, and 10th left ribs. Pale cyst-like areas in right tibia and fifth left metacarpal, with no expansion of corticalis. Calvaria shows innumerable pale rounded mottled shadows many of them more than 1 cm. in diameter (Fig. 200). Greatly deformed tri-radiate pelvis with fracture of superior ramus of os pubis on each side (Fig. 202). In a lateral view the thoracic and lumbar vertebrae show



FIG. 200.—*Case 9.* Osteomalacia. Radiogram of skull before treatment.



FIG. 201.—*Case 9.* Radiogram of skull after treatment.

considerable bi-concave deformity. By contrast with the bones of a control subject the right femur, tibia, humerus, radius, ulna, and hand, as well as other bones, show definite diminution in density (*Fig. 199*).

Radiograms of renal tract show no calculi.

*Calcium and Phosphorus Metabolism.*—Observations were made upon the blood chemistry at intervals over a period of eleven months. Before treatment the serum calcium varied between 9.6 and 10.1 mgrm. per 100 c.c., and the plasma



*Fig. 202.*—*Case 9.* Osteomalacia. Radiogram of pelvis.

phosphorus was constantly 1.5 mgrm. per 100 c.c. Plasma phosphatase 1.2 mgrm. The calcium output was estimated in the urine and faeces for three three-day periods, the patient being kept on a weighed diet of known low calcium content. The calcium output in urine and faeces was slightly less than in the control (*Fig. 180*).

*PROGRESS.*—A portion of bone was removed for histological section.

*OPERATION* (Mr. R. Milne, July 7, 1930).—A piece of bone was removed from the inner aspect of the right tibia. Periosteum, corticalis, and a portion of spongiosa were included.

#### Pathological Report.

(PROFESSOR H. M. TURNBULL.)

Tissue removed at operation. (S.D. 1573/1930.)

#### Tibia.—

*MACROSCOPIC.*—The specimen was a longitudinal slice from the inner aspect of the tibia. It was 10.5 cm. long, 0.5 cm. deep at each end, and 1 cm. deep in the centre. Beneath the periosteum was an apparently homogeneous corticalis, about

0.25 cm. deep, and beneath this a spongiosa of thin trabeculae. The spongiosa contained pale yellow, almost white marrow.

**MICROSCOPIC** (*Fig. 195*).—The bone was divided into three pieces and these were decalcified in 4 per cent formaldehyde in distilled water, Müller's fluid, and, finally, nitric acid. The decalcification is not complete. In large areas Kossa's reaction is positive. A control is thus obtained to the estimation by hæmatoxylin-staining of previous calcification in other areas. The periosteal surface is indented by numerous bays, some of which communicate with medullary spaces. The outer fifth of the bone is relatively compact, but has a spongy structure (*Fig. 195*) owing to numerous medullary spaces, which are directed for the most part in the long axis of the bone. There are very few Haversian canals; such as are present are usually wide. In the inner four-fifths the medullary spaces are much larger, so that the bone forms a spongiosa of wide mesh. There are a few small external fundamental systems and several interstitial systems of woven bone; all the rest of the bone is lamellar. There is an osteoid zone on almost all the free surfaces of the bone. Some zones are very narrow, but most are abnormally conspicuous. They are very wide where they have been cut tangentially. Measurements of those that appear to have been cut at right angles to their surfaces show that many are twice as deep as the deeper zones in the bones of healthy infants. Others are much deeper. Some are shown by embedded cells to be composed of two or even three lamellae. There are also still deeper osteoid zones which in places show ill-defined patches of granular calcareous impregnation. Such patches of partial calcification usually lie in the osteoid zone where it meets the fully calcified bone, but they sometimes lie nearer its centre. The osteoblasts form a single line of narrow cells with very scanty cytoplasm, and they are frequently at a wide distance from one another. In these respects they appear to be less active than in normal infants. An osteoclast in a Howship's lacuna was found after a search through several sections. Some of the osteoid zones lie upon surfaces showing lacunar resorption; they are more often separated from such surfaces by a calcified lamellar system of which they form part. The shallower subperiosteal bays contain a cellular fibrous marrow. All the other spaces contain an adipose marrow, which contains a few hæmatogenous cells in one or two places.

**Remarks.**—Osteoid zones are present on almost all surfaces. They are exceptionally numerous and exceptionally deep for a healthy adult. Many are twice as deep as the zones in the active growth of early childhood; others are much deeper. Many of the zones are, therefore, deeper than those in the examples of osteitis fibrosa already described, in which the depth was within the limits measured in healing fractures. In osteitis fibrosa and healing fractures the increase in depth beyond that in healthy infants depends upon the greater rapidity of apposition. Rapidity of apposition can be gauged approximately by the size and number of the osteoblasts. If there were very numerous and large osteoblasts upon the zones in the present case, it would be difficult to be certain that the unusual depth was not due to unusual rapidity of apposition, because the depth is seldom conspicuously greater than in healing fractures. The number and shape of the osteoblasts, however, shows that apposition is proceeding very slowly or is in abeyance. The abnormal depth of the osteoid zones and their occasional patchy and powdery impregnation with calcium salts are due, therefore, to complete and partial cessation of their calcification, though a chemical removal of calcium salts from them cannot be excluded on purely histological grounds. In either case the abnormally deep osteoid zones here are definite evidence of osteomalacia.

The osteomalacia is associated with great osteoporosis. As no skeletal



deformities were noted in the childhood of the patient it can be assumed that a compact corticalis was formed in due course, and that the present porosity is due to lacunar resorption. The perfect lamellar structure of the bone that remains suggests that a compact corticalis had been present in adult years. There is extremely little evidence of lacunar resorption at the time of removal of the specimen: only one osteoclast in a Howship's lacuna was found on searching several sections. Surfaces eroded by lacunar resorption are either covered by an osteoid zone, or, more often, by an osseous lamellar system continued by an osteoid zone. Much of the resorption, therefore, has preceded the present attack of osteomalacia.—H. M. T.

**SUBSEQUENT PROGRESS.**—The patient was given a diet of high calcium content, together with 15 grm. of calcium lactate and two tablets of radiostol (British Drug Houses) daily. She was sent home to continue this treatment. Two months after commencing it she became pregnant. She then continued to take the high calcium diet and the calcium lactate but reduced the daily dose of radiostol to one tablet. In spite of the pregnancy and of morning sickness she made rapid clinical improvement. Within three months the pains disappeared from the bones and she began to walk. Within six months she was able to get about the house, to climb the stairs unaided, and to do many household duties such as ironing and baking bread. At this time she was re-admitted to hospital for further investigation. The clinical improvement was fully borne out by the physical examination. Radiograms of the bones (Jan. 13, 1931) showed union of many of the fractures and complete healing of the defects in the calvaria (*Fig. 201*). In spite of these facts the plasma phosphorus was still constantly low, namely, 1.0 mgrm. per 100 c.c. Serum calcium 10.9 mgrm. per 100 c.c. Plasma phosphatase 0.44 mgrm. The dose of radiostol was increased to three tablets daily and ultra-violet irradiation was commenced. Within four weeks the serum calcium rose to 13.7 mgrm. per 100 c.c., but the plasma phosphorus remained low, namely, 1.8 mgrm. per 100 c.c. The patient was sent home on a high calcium diet without radiostol. Except for morning vomiting she remained well, free from pain and able to do her housework. She was finally re-admitted eleven months after treatment was first commenced. At this time the pregnancy was approaching full term. The serum calcium was 13 mgrm. per 100 c.c. In the thirty-sixth week of pregnancy Cæsarean section was performed, together with sterilization by partial excision of the Fallopian tubes. A live female child weighing 5 lb. 4 oz. was delivered. At the operation the pelvic measurements were taken.

Symphysis pubis to promontory 5.9 cm.

Right ileopectineal line to promontory 3.8 cm.

Transverse diameter at brim 9.5 cm.

Distance between nearest points on ileopectineal lines 3.8 cm.

The mother remained well, the low plasma phosphorus and high serum calcium persisting. The baby gained weight, and controlled radiograms of its bones showed no evidence of rickets.

**Remarks.**—This case is one of 'low phosphorus' osteomalacia. The liability to spontaneous fractures in this type of the disease has been pointed out by Maxwell (1929). The diagnosis is based on the clinical picture, the histology of the bone, the blood chemistry, and the calcium balance. Over a long period the serum calcium was high and the plasma phosphorus low. Under such circumstances confusion with hyperparathyroidism is avoided by determining the calcium balance, thereby demonstrating the absence of an increased excretion of calcium. Confusion may equally be avoided by removing a portion of bone for histological section. The remarkable clinical and

radiological improvement in spite of the pregnancy shows that the absence of vitamin D must be a potent factor in the causation of osteomalacia. It is not necessarily the sole factor.

For his inspiration and example no less than his expert collaboration in pathology I have to thank a great master, Professor H. M. Turnbull. It is also a pleasure to thank Miss Simmonds and her staff for their valuable work both in nursing and dietetics, and I am indebted to Mr. A. J. Walton for the courage and skill of his operative technique. I most gratefully acknowledge the assistance received from Dr. F. B. Byrom in carrying out the animal experiments. My thanks are due to at least fifty other friends and colleagues, only a few of whom are mentioned in the text.

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## THE SURGICAL TREATMENT OF PARATHYROID TUMOURS.

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ONLY a few years ago the anatomy of the apparently insignificant parathyroid glands was hardly known, and in fact many believed that they consisted only of misplaced portions of thyroid tissue or of small lymphatic glands. The appreciation of the fact that injury of these bodies in the operation of partial thyroidectomy might lead to the onset of tetany has of recent years directed considerable attention to their exact situation. More extensive work upon calcium metabolism, of which that of Dr. Donald Hunter in this country is so brilliant an example, has demanded even more detailed knowledge of their normal and abnormal situations. The glands are so small and so hidden away that the diagnosis of a parathyroid tumour depends almost entirely upon indirect evidence. In all four of our cases there were no physical signs of a tumour in the neck. The careful investigations of Dr. Hunter upon the bone changes and upon the calcium and phosphorous metabolism enabled him, however, to say dogmatically that a tumour was present. This becomes a matter of great importance, for in one of our cases the tumour could not be found at the first operation, and I am certain that in a not inconsiderable number a similar failure has occurred elsewhere. Fortunately in our case Dr. Hunter was so certain of the presence of a tumour that a second operation was undertaken, whereby not only was it found but knowledge was obtained which I hope will be sufficient to prevent similar tumours being overlooked in the future.

The number and position of the parathyroids is variable, but as a general rule there is a superior and inferior body on each side, although in many cases these may be increased to three or four. The wide variation of the position of these bodies is shown in Halstead's figure, based upon the investigation of 67 cases of McCullum, which is reproduced in de Quervain's work on goitre. This shows that they occupy a relatively wide strip on the posterior border of the thyroid gland extending from the upper to the lower poles. According to de Quervain they lie in this position between the thyroid capsule and the thyroid fascia, a position which is clearly represented in de Quervain's diagram. The superior bodies may be placed somewhat far forward, and receive, according to Curtis, their blood-supply from branches of the superior thyroid artery. Lahey, who states that one or more parathyroids are not infrequently removed during the operation of partial thyroidectomy, finds that it is on the upper pole especially that this accident is likely to happen. In all my own cases, and in all the post-mortem specimens that I have been able to examine, the superior bodies, although variable in their

lateral and vertical positions, have always been placed anterior to the thyroid fascia and between it and the thyroid capsule.

The inferior glands are placed in close relationship to the inferior thyroid artery, from which—also according to Curtis—they obtain their blood-supply.

They may be placed at a very varying level, and in their lower positions come into close relationship with the inferior thyroid veins, where they are more liable to injury. They are usually, like the superior bodies, described as lying inside the thyroid fascia. Our experience has shown, however, that there is here a very important variation, a variation which determines the path of spread of a tumour. They may be situated below the artery, in which case they are usually anterior to the fascia (Figs. 203, 204, Position I), or above the artery, when they are often deep to the fascia, being in this case only visible from the posterior surface or after division in the fascia (Figs. 203, 204, Position II). This latter position, as far as I know, has not been previously described, although in one of the figures in Dunhill's paper it appears to be depicted in a position behind this fascia. As, however, this variation may lead to a tumour's occupying a most unexpected position, it is, we believe, the most important variation that may occur.

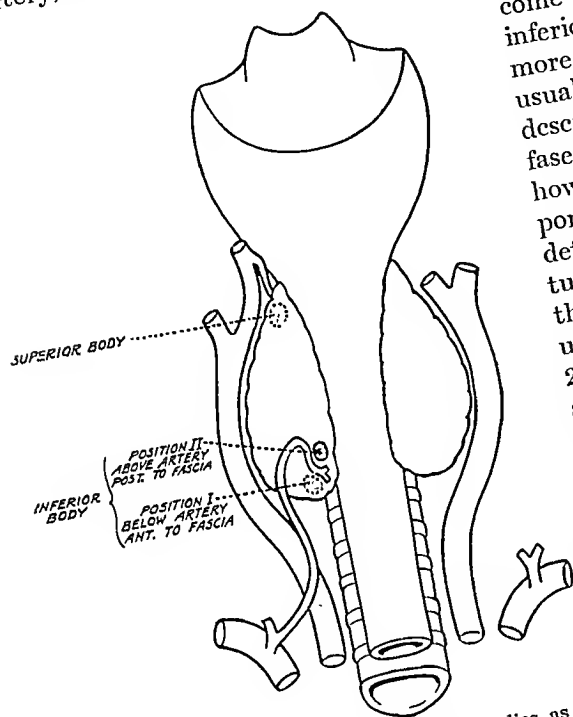


FIG. 203.—Position of parathyroid bodies as seen from behind.

tion, it is, we believe, the most important variation that may occur.

Although the above may be considered as the more or less normal positions, there are other abnormalities which are important to remember. Occasionally one or more parathyroids are situated in the substance of the thyroid gland (Fig. 204, Position III). This arrangement, said to be normal in the dog, is rare, but is, however, of surgical importance in that the gland is certain to be removed in a partial thyroidectomy, as in a case reported by Lahey. Also with an expected parathyroid tumour great difficulty may arise if a small adenoma of the thyroid is present; for this may be mistaken for the parathyroid tumour in the substance of the gland, and the true tumour thereby thyroids may exist anywhere in the branchial area, and apparently even in

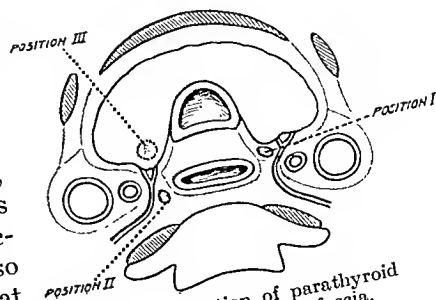


FIG. 204.—Relation of parathyroid bodies to the thyroid fascia.

the substance of the thymus, but I can find no account of a parathyroid tumour being so situated.

Tumours of the parathyroid have been regarded as very rare, and certainly relatively few operations have been performed for their removal. Compere, in 1930, was able to collect only 12 cases in addition to one of his own. It was only in 1907 that Erdheim propounded his view that enlargements of the parathyroid with certain bone disease were an attempt on the part of the organism to compensate for a loss of calcium, and thus were the result rather than a cause of the disease. It was not until 1926 that Mandl, in testing this theory, found that the bone symptoms were increased by parathyroid grafting, and acting on this observation found a tumour behind the left lobe of the thyroid which he removed together with the parathyroids which he had previously grafted into the abdominal wall. The removal of the tumour, which proved to be a parathyroid adenoma, was followed by marked relief.

In this country the recent work of Dr. Hunter has directed considerable attention to these bone changes, and it is very probable that the disease will prove to be much more common than was at one time thought. Since the publication of his first paper he has had four cases in which he has been able to make a positive diagnosis of parathyroid tumour—a diagnosis which I was able to confirm at operation. Although these cases have been so relatively few in number, the operation has brought to light certain difficulties in the technique, and has shown how they may be avoided.

In all cases a light ether anaesthesia was used, and it was realized that a wide exposure was necessary, for since in no case could a tumour be felt it was important to examine not only the normal sites of all the parathyroids, but it was soon realized that the tumour might be situated at some distance from its expected site. A wide collar incision was therefore made, the sternomastoids were freed and fully retracted, and the pretracheal muscles divided transversely in one sheet at the level of the isthmus of the gland. By widely dissecting the flaps thus formed upwards and downwards the whole of the thyroid gland is freely exposed from the upper to the lower poles. This gives a much wider exposure than a vertical split in the pretracheal muscles, and if they are carefully sutured afterwards there is no resulting deformity or weakness.

The thyroid fascia is incised, the lateral lobes of the gland are freed and rolled inwards in turn. To accomplish this fully it may be necessary to ligature and divide the middle thyroid vein. By this means a tumour may be at once discovered in the usual site of the parathyroid body, either superior or inferior. In our first case (*see below*) a tumour was thus found in the normal site of the left inferior parathyroid body and was easily removed. In no one of our cases so far has a tumour been situated in the region of the superior parathyroid bodies. Experience of later cases has shown, however, that the real difficulties arose according to the relation of the inferior bodies to the thyroid fascia. If the parathyroid body lies between this structure and the capsule of the thyroid gland (*see Figs. 203, 204, Position I*), it will as it enlarges pass downwards along the inferior thyroid veins and in front of the carotid artery in a manner identical with a retrosternal goitre until it comes

to lie between the sternum and clavicle (*see Fig. 206, Position I*). Hence if no tumour is observed in the usual site, a finger should be passed into the thorax on either side of the trachea. In our third case a little confusion was at first caused by the fact that a small adenoma was present in the left lobe of the thyroid which might have been a tumour in an aberrant parathyroid, but on passing a finger down alongside the trachea a large tumour was at once found in the upper part of the thorax on the left of the trachea, and although it measured about  $3 \times 1$  in. it was easily dislocated and removed, leaving a large cavity immediately behind the sternum exactly like that left after the removal of a retrosternal goitre. Very different is the condition if the parathyroid gland is situated behind the thyroid fascia (*see Figs. 203, 204, Position II*), for when the fascia is opened from the front no sign of the tumour is seen and nothing can be felt in the thorax on examining either side of the trachea.

In our second case no tumour could be found in the usual situation, but on making a small incision through the fascia above the inferior thyroid artery (*Fig. 205*), a tumour about the size of an almond was immediately found and removed. The importance of this relationship was not, however, fully realized at the time, for the tumour being small had not passed from its normal position.

The position of the tumour in our fourth case brought to light a very important relationship which I believe has not been previously described, and probably explains those cases in which a tumour has not been found at operation. In this case an investigation of the normal sites revealed no tumour, and as a small mass was found in the lower part of the right lobe of the thyroid it was removed in the belief that it might be a parathyroid tumour

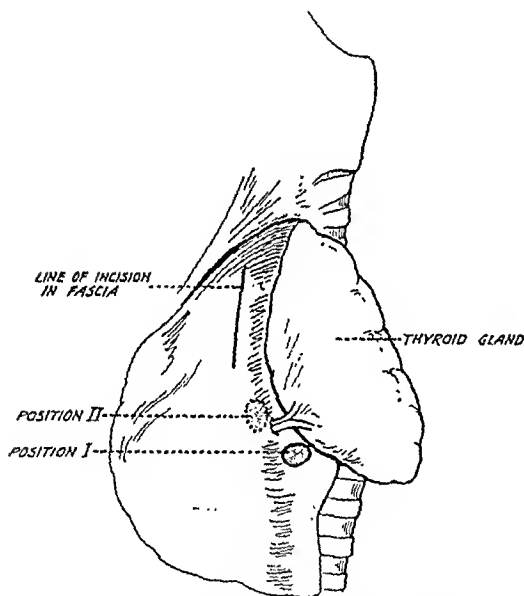


FIG. 205.—Incision in thyroid fascia to expose a deep tumour of the inferior parathyroid body.

lying within the thyroid gland. This mistake should not have been made, for Professor Turnbull, who was present at the operation, was convinced, from the colloid appearance of the mass, that it was an adenoma. Sections after the operation confirmed this view, and as Dr. Hunter was certain that a parathyroid tumour was present a second operation was performed five days later. By this time a study of the anatomical relationships had suggested what was later found to be a fact. Therefore when a second investigation had confirmed the view that no tumour was present in the usual sites a small incision was made in the thyroid fascia (*Fig. 205*). A small typical parathyroid tumour was at once found behind the oesophagus and removed, but as a wide space was then revealed a finger was inserted and a large tumour,

as large as a plum, kidney-coloured, was found lying immediately in front of the 2nd and 3rd dorsal vertebra. It was easily dislocated upwards, and its vascular pedicle, which ran from the inferior thyroid artery, was ligatured and the tumour removed. The position of this tumour made it evident that an enlargement of the inferior parathyroid body which is situated behind the fascia passes downwards behind the œsophagus immediately in front of the vertebrae until it comes to lie in the thorax and is quite out of sight (*Fig. 206, Position II*). In this case, as in *Case 2*, the large cavities were drained with a small tube, the muscles were approximated with catgut, and the skin was united with fine sutures. The surgical after-treatment was in no way different from that of a large adenoma of the thyroid, but careful attention had to be given to an estimation of the blood-calcium, and the possibility of the onset of tetany guarded against by the administration of calcium and parathormone. The details of the after-care in this respect have been fully described by Dr. Hunter, and therefore need not be repeated here. The full details of the cases have also been reported by Dr. Hunter (*see p. 213*), and therefore need only be briefly summarized.

### CASE REPORTS.

*Case 1.*—M. M., female, aged 41. For five years pains in joints and limbs. Diagnosed as osteoarthritis. Five months before admission, X-rayed and diagnosed as osteitis fibrosa. One month before, fracture of neck of left femur. On examination marked hypotonicity of muscles, shortening and eversion of left lower limb, tenderness on pressure over shafts of long bones, tender swelling in lower end of right ulna. X rays showed general osteoporosis and multiple cyst-like areas in right ulna, right tibia, right patella, and phalanges. Increase of serum calcium and a low plasma phosphorus. No tumour felt in neck. Operation on Nov. 29, 1929. Tumour removed from region of left inferior parathyroid. Good recovery and great improvement of all symptoms.

*Case 2.*—M. R., female, aged 37. Two years pain in knees, hips, heels, and arms. Weakness steadily increasing. For two years unable to get about. Swelling of right lower jaw. Limbs becoming stiff and distorted. Swelling of left forearm and right hand. In many hospitals since. In May, 1930, admitted to the London Hospital. Much deformity. Height 4 ft. 10 in. In bed totally disabled. Hips and knees flexed, cannot move either arm. Great pain in limbs. Bones very tender. All long bones much distorted and bent. Large tumours right lower jaw,

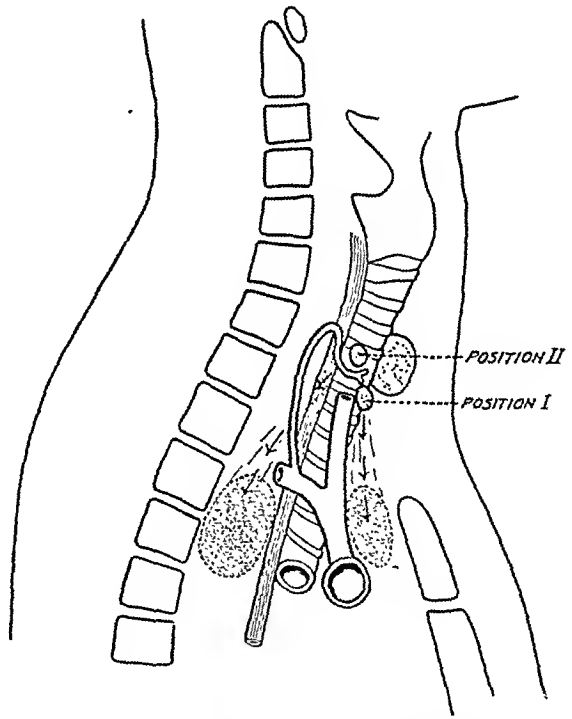


FIG. 206.—Alternative positions of the inferior parathyroid bodies.



dorsum of right hand, and second metacarpal. General osteoporosis. Heads and necks of both femora absent. All long bones thin and much bent. Calculi in right kidney. Serum calcium increased. No tumour in neck. Operation on July 2, 1930. Small tumour  $\frac{1}{4} \times \frac{1}{2}$  in. removed from behind thyroid fascia at site of right inferior parathyroid. Great improvement since operation.

*Case 3.*—A. F., female, aged 40. During pregnancy twelve years ago difficulty in walking. Easily tired. Child stillborn. Two years ago pain in joints; interfered with walking. Two and a half years, swelling in right tibia, portion removed. Severe pain in right leg since. Swellings developed on left ring-finger, forehead, and chin; varied in size from time to time. In bed owing to pain in joints two years. Some frequency of micturition. Six years ago stone removed from left kidney. Small swelling felt in left lower pole of thyroid. Hard, bony tumours in lower right tibia, skull, left ring-finger. General osteoporosis. Serum calcium increased. Low plasma phosphorus. Albumin in urine; right and left renal calculi. Operation on Aug. 29, 1930. Small adenoma felt in lower left lobe. Large rounded tumour  $3 \times 1$  in. in thorax behind sternum and to left of trachea; easily dislocated upwards and removed. Wound drained and closed. Patient recovered well from operation and has made great improvement since.

*Case 4.*—B. Y., female, aged 51. At the age of 13 abscess of left hip, probably osteomyelitis. Operation on right arm and left hip at the ages of 14 and 15. Commencing deafness at 16 (1915). Swelling of outer side of right orbit. March, 1926, swelling on right gum with stiff jaw. October, 1926, operation on right jaw. August, 1927, swelling on left side of nose. December, 1927, myelomatous tissue removed from left side of nose. In 1928 general tenderness over bones; swelling in left thigh. June, 1928, deep X-ray treatment. December, 1927, could hardly lift herself in bed. Large swelling in left femur, smaller swellings in right femur and right tibia. Deafness getting worse. Trace of albumin in urine for some years. On examination very thin. Bony swelling on outer aspect of right orbit. Sars over upper end both femora and right humerus. Swelling in middle of right tibia. No marked deformity. General osteoporosis; little bending of right radius. Cystic spaces in upper end of right humerus, upper end of right femur, lower end of right femur, and upper end of right tibia. Thyroid not enlarged; no tumour felt in neck. Urine contains one-twentieth albumin. Small renal calculi. Increase in serum calcium and decrease of plasma-phosphorus. Operation on Nov. 26, 1930. No tumour in normal parathyroid regions. Both superior and left inferior parathyroids identified in their normal positions. Small tumour removed from lower right lobe. Good deal of vomiting after operation. Sections showed tumour to be adenoma of thyroid and the blood calcium was unaltered. Second operation on Dec. 1, 1930. Old wound opened. Incision on right side through thyroid fascia above inferior thyroid artery. Small tumour  $\frac{1}{2}$  in. long found behind œsophagus. Large tumour felt low down in thorax immediately on dorsal vertebræ and behind œsophagus; dislocated upwards; found to be the size of a plum and kidney-coloured. Pedicle running from it to inferior thyroid artery ligatured and divided. After this operation there was no more vomiting, the patient rapidly improved, and the serum calcium fell. She has made very great improvement since operation.

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## ‘MADE-TO-ORDER’ SPLINTS: THE TECHNIQUE OF CONSTRUCTION AND FIELD OF APPLICATION.

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IN the treatment of fractures external supports or splints play a leading rôle. Many, if not most, of the splints still figuring in surgical text-books do not provide adequate and comfortable support for the injured member, and some are frankly preposterous. The ideal splint for any part should be efficient and comfortable to wear, easy to make and to apply; should fit snugly on to a large surface so that it may not move in relation to the patient no matter how he may twist and wriggle; and, lastly, it should be of small cost and easily procurable anywhere at any time. It is obvious that these requirements cannot be met by splints of a fixed shape such as those of metal or wood. These require the skill of a trained surgeon for their application, and only too often are out of gear before the patient has reached the front door.

Suppose that a tailor made suits of clothes to a certain fixed shape, and in order to adapt them to different customers introduced voluminous pads here and there to fill up dead spaces! Even supposing that the pads did not shift, the customers would probably take no great pride in their apparel. Nor would a dentist attain to much popularity if he constructed tooth plates to a standard design. Yet the wooden and metal splints in use in the world of surgery do not and cannot fit, and necessitate the introduction of such substances as cotton-wool, felt, and so on.

It is not the intention of the author to discuss the treatment of fractures, arthritis, and so on, but simply to describe a method of constructing plaster splints. In the first place consideration will be given to certain principles which apply in general, and in the first section reference will be made to the materials used and certain points of importance in the manipulation of plaster-of-Paris. Later the construction of certain splints will be described. These are to be regarded as samples only. The breadth of the field of usefulness of the method is proportional to the amount of ingenuity possessed by the surgeon.

### GENERAL CONSIDERATIONS.

The best form of fixation for the fragments of a broken bone would be a closely-fitting ferrule, such as is used to unite the segments of a fishing rod, but this is out of the question as the bones are surrounded by soft parts. It is, however, absurd to introduce *more* soft and yielding material between skin and splint, and in general it may be stated that the more closely the splint approaches the bone fragments to be supported the better will be the fixation. Therefore, it is desirable that the apparatus should fit directly and snugly on

to the skin of the part. Now circular plasters could be made in this way, but for many reasons which need not be discussed these are unsatisfactory and often dangerous. It will be seen later that most of the splints described are more or less semi-cylindrical or crescentic in section. Thus ample provision is made to permit of swelling of traumatized tissues and ensure against constriction of the part. It may be stated dogmatically that a well-fitting plaster support never causes the least damage to healthy skin, either on account of its chemical constitution or its physical characteristics; and, conversely, that sores are always the result of errors in technique.

The snug fit, in addition to ensuring the maximum of efficiency, carries with it other advantages. In many instances cotton-wool and similar substances may be dispensed with entirely, and in some (e.g., the humeral and clavicular supports) no bandages are required. Removal is easy and re-application is almost fool-proof. Thus when the time comes for voluntary movement, massage, and so on, the management of the splint may be left safely to any intelligent individual, with consequent great saving in time to the surgeon. Inspection of the uncovered portion of the limb is easy without removal of the plaster at all.

#### Materials Used.—

*Plaster-of-Paris.*—It is not necessary to pay high prices for special grades. Any plaster which will set satisfactorily in from ten to fifteen minutes will do.

*Scrim, or Hessian.*—This is made in various weights. It is better to use scrim with a wide mesh, as this adapts itself well to surfaces of irregular contour and is easily penetrated by the plaster cream. 'Eight-ounce scrim' is the most satisfactory and may be obtained at any decorators. As a fabric it is much cheaper and far more serviceable than the ordinarily used leno.

*Gauze*, in rolls one yard wide.

*Webbing Strips.*—These are obtainable in various widths and should be stout and inextensible. The most useful widths are one inch and two inches.

*Buckles* with sharply pointed prongs in sizes to fit the strips to be used.

In addition to the above all sorts of odds and ends are at times incorporated in the plasters at the whim of the surgeon. It is the practice of the author to carry in his car a supply of the above-mentioned materials, with the result that at any time or place he can manufacture a really efficient splint. The saving in time and trouble which results from this simple provision is considerable.

**Garb of the Operator.**—It is well to prepare for the worst. The surgeon should wear a gown, canvas leg covers like waders, and rubber gloves. The skilled manipulator of plaster does not, however, make much mess.

### THE MAKING OF PLASTER SPLINTS.

The method to be described is very easy, but success depends on attention to detail, and in particular it is important to have everything in readiness before mixing the plaster cream. First of all the materials to be used must be prepared. A strip of gauze is cut measuring twice the width of the area to be covered and as a rule at least from six to twelve inches longer. There is no need to shape the gauze. Too small a piece will cause trouble later,

whilst excess is easily cut away when the time comes. Strips of scrim roughly corresponding to the shape of the proposed splint, but always a good deal smaller in all directions, are then cut out. For very small plasters about three or four will suffice; for plaster beds about a dozen or more should be prepared. Small scraps of scrim may be used to insert where extra thickness is necessary. Strips of webbing of the appropriate length should be cut. Buckles are affixed when the splint is removed for trimming. If it is proposed to use wires, metal strips, and so on, these must be to hand and shaped appropriately. When all the materials to be used are ready attention is transferred to the patient.

Infinite pains must be taken to place the part in question in the required position, and here gentleness and patience avail more than anything else. It is useless to make a support on a limb in the wrong attitude, for once this is done there is no means of altering the state of affairs. The surface on which the slab is to be laid is arranged so as to face upwards if possible, and sand-bags or other objects are placed where necessary so that the part is supported firmly in the correct alinement. The preliminary posing of the part is the most important detail in the manufacture of these splints, and no amount of skill in the carrying out of other manipulations will compensate for shortcomings here. An anæsthetic may be given to attain the desired result, but this is not necessary as a rule.

From this point onwards until the completion of the process there should not be the slightest disturbance of the injured part. The advantages of this method when compared with others, which frequently enough are attended by movement, pain, and consequent spasm, should be obvious.

When the surgeon is satisfied with the position of the patient the piece of gauze is laid over the surface in question with its edges projecting all round. It is then time to mix the plaster cream. A bowl of convenient size is taken and the required amount of water with the chill off is poured in. Plaster is then dusted on to the surface of the water so that it is wet as it falls in. Enough is added to produce a cream which will not easily run out of the meshes of the scrim, but which is thin enough to penetrate thoroughly the strands of the fabric. The bowl of cream and other materials are then placed a few inches only from the seat of operations. This is important, as thereby much time is saved and the amount of plaster dropped in transit from bowl to splint is minimized and localized to a small area. A little cream is next smeared on the gauze where it is in contact with the skin to ensure that it will be properly incorporated in the finished article. A strip of scrim is taken, laid dry on the appropriate area, and immediately smeared over with plaster cream, care being taken to fill all the interstices of the fabric to the exclusion of air. Other strips are then added in a similar way until the required thickness is attained. The reader will remember that the strips were cut smaller than the area to be covered. They are placed so that whilst they overlap along the middle line of the splint, alternate layers only reach to one or other edge. Thus thick clumsy margins are avoided and, without sacrificing strength, weight is cut down. Reference to *Fig. 207* will make this arrangement clear. Any ragged tags or corners are cut away with scissors as each layer is placed in position. If any webbing bands are to be incorporated,

these are laid at the appropriate positions between two of the layers of scrim and are thus firmly welded into the plaster.

When the requisite thickness is attained the margins of the gauze projecting beyond the splint edges are turned back over the surface of the latter and cemented down with a little of the cream. By a process of wiping the plaster away from the margins into the body of the structure a neat thin edge bound by the reflected gauze layer is obtained. The gauze must be cut in order to permit of this turning back where webbing strips emerge. As the setting process begins the surface of the plaster is smoothed over by the gloved hand, which is frequently rinsed in water. When set the plaster structure is removed for inspection and the patient's skin may be cleaned. The splint is then re-applied and fixed in position by bandages or webbing bands as the case may be. A single layer of very thin material such as silk or gauze may be worn between skin and plaster.

All of the splints to be described are made as outlined above, but there are certain other points to which attention must be drawn. When the area on which the slab is to be constructed is hairy it is wise to smooth down the hair with a thin smear of oil or vaseline. Shaving is unnecessary. If the splint is removed as soon as it has set, such hairs as have become caught in its surface are easily freed without causing pain. When making plaster

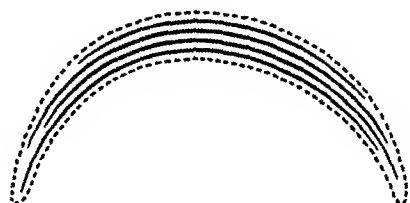


FIG. 20—representation of a plaster splint, to show the following points. The plaster extends about half-way round the circumference of the limb. Strips of scrim are so disposed that at the centre of the splint there are many layers, at the edges one. Thus a bevelled or crescentic effect making for neatness and lightness combined with strength is obtained. The interrupted line represents the enveloping layer of gauze which serves to bind the edges as well as to hide the coarse meshes of the scrim.

beds and splints such as those described for the treatment of fractures of the clavicle and humerus precautions are taken so that the skin over prominent bony points may be guarded from trauma. Before applying the gauze a disc of cotton-wool, measuring 2 or 3 in. in diameter and about  $\frac{3}{8}$  in. in thickness at its centre and fading away to the periphery, is prepared and placed directly on the skin area in question, after wetting the latter with water so that slipping of the wool during subsequent manipulations is hindered. The wool is removed from the finished plaster and thus a shallow depression in the surface is provided for the reception of the bony prominence. Should the splint press upon or rub the skin at any point, causing pain, the tender spot is marked with ink, the splint is re-applied and on removal the offending part of its surface is seen to be plainly marked. The plaster is then scraped away so that contact with the skin is eliminated. The surgeon should always aim at providing a shallow, shelving trough rather than a sharply defined hole for the reception of prominent points.

When there is a wound or ulcer in the region of the part to be splinted matters may usually be arranged so that the apparatus is made on the healthy aspect of the limb. Dressing of the wound is thus easy. If, however, it is necessary to make the splint on the affected aspect of the part in question, a dressing should be fixed in position over the wound or ulcer and then

covered with a layer of oiled silk or other waterproof material. The splint is then constructed over all, and when finished contains a recess for the accommodation of the dressing. It is not necessary to bandage the dressing as it is held firmly in position by the applied splint.

Sometimes it is desirable to coat over the plaster apparatus with shellac dissolved in methylated spirits. Several coats may be applied with an ordinary varnish brush. The resulting surface is hard, smooth, durable, and waterproof, and may thus be washed. Plaster beds and other splints which are designed to serve for more than a short period are, as a rule, treated in this way.

In order that the splint may be strong and yet light and neat in appearance, the following points should be observed. Tubular structures are immensely strong, and to a less degree so are semi-cylindrical forms. Hence the plaster should be constructed so as to approach the latter form wherever possible. When the curve is small or absent the segment in question must be made much thicker to compensate for the inevitable loss of strength. Thus in a plaster bed the interseapular region is flattish and must be made very thick. Weight is reduced and neatness in appearance attained by bevelling so that the splint diminishes in thickness from the centre in all directions to the margins. It is never necessary to incorporate metal in order to augment strength.

#### INDIVIDUAL SPLINTS.

In this section it is proposed to deal briefly with some of the more important points arising in connection with the construction of some useful splints. Surgeons who have the necessary ingenuity and the will to do so will have no difficulty in designing and constructing apparatus to meet almost every conceivable contingency.

**Plaster Beds** (*Figs. 208-210*).—The patient lies prone with a pillow under the pelvis and upper chest, and a block supporting the forehead. The arms are abducted, forming with the body an angle of not more than  $20^\circ$ . Bony prominences are cared for as outlined above. It is wise also to fill up deep grooves, such as that over the spines of the lumbar vertebrae between well-developed erector spinae muscles, with wool, in order to avoid prominent ridges on the bed surface. The bed is shaped like the posterior half of a waistcoat, but extends well on to the buttocks and reaches always to the base of the neck, and, if the lesion is above the 6th dorsal vertebra, to the occiput or further. Laterally the edges reach to the mid-axillary line. It is necessary to make the apparatus especially thick in the seapular region to guard against breaking. Often a large 'hump' of scrim and plaster is built on the dorsal aspect in the lower lumbar area. This raises the lower edge from the bed and facilitates the use of the bed-pan. It is better to make the plaster bed too heavy rather than too light. For an adult about 18 or 20 lb. of plaster should be used, but the amount varies of course with the bulk of the patient. The surface is usually coated with shellac varnish. A well-made plaster bed properly used for years and provides a very efficient and comfortable support for the trunk. The cost of materials used is about five or six shillings.

A very satisfactory apparatus (*Figs. 211, 212*) for the treatment of arthritis of the hip and some fractures of the femur (where extension is not needed), and for supporting the limb after operations such as grafting an

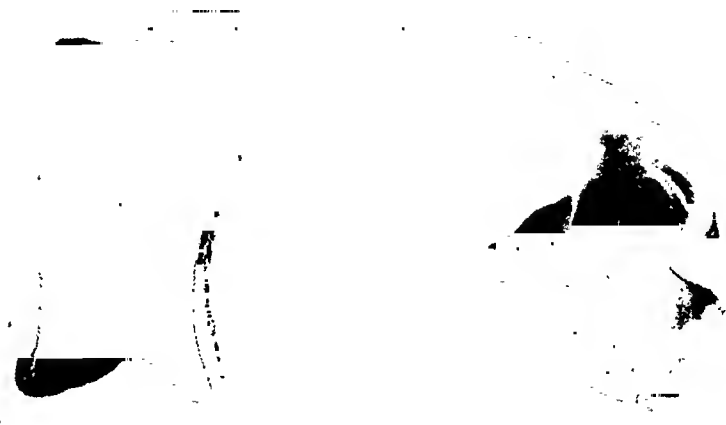


FIG. 208-210.—A plaster bed for use in the treatment of injuries and diseases of the spine. Note the pronounced lateral projections in the pelvic, thoracic, and neck regions; and the hump, which permits of see-sawing with but little effort on the part of the nurse. *Fig. 210* shows the buttocks raised to facilitate the use of a bed-pan, a support having been removed from the shoulder region.



un-united fracture of the femur, may be made by extending a plaster bed down to and including the foot. Similarly, an extension may be provided to support the arm in a position of wide abduction at the shoulder. If such splints are made prior to operation, a dummy dressing should be placed in



FIG. 211, 212.—A plaster bed with leg extension. This splint was made prior to operation for fixation of the hip-joint. A dummy dressing was applied over the site of operation in order that there should be a recess in the plaster surface for the accommodation of the post-operative dressing. Fixation is very efficient and transportation easy.

position on the limb in order to provide a recess for the accommodation of the post-operative dressing.

**Apparatus Used in the Treatment of Fracture of the Clavicle (*Figs. 213-215*).**—Two plaster slabs are made, one on the sound shoulder, the other on the elbow region of the affected limb. The first is to carry the weight of the injured limb and apparatus and is made on the patient in the upright position.



It is oval in shape, reaching to the clavicle in front and well on to the back behind. A strip of webbing with a buckle at each end is incorporated. The second slab is moulded over the elbow region of the injured limb whilst the patient lies supine with his injured shoulder thrust cranially and his hand placed well over his sound shoulder. A flange about two inches wide fits on to the thorax. The bony points round the elbow are provided for as outlined in the previous section. A webbing band is incorporated to link up with the weight-carrying slab. The provision of two buckles makes adjustment easy, and the limb carrying the outer fragment is supported so that the shoulder is a good deal higher than that on the uninjured side. The elbow region is firmly fixed, and the weight of the limb tends to carry the shoulder backwards and outwards with this fixed point as a centre of rotation. This movement almost always brings the fragments into the required position. The apparatus, when properly applied, can scarcely get out of adjustment,

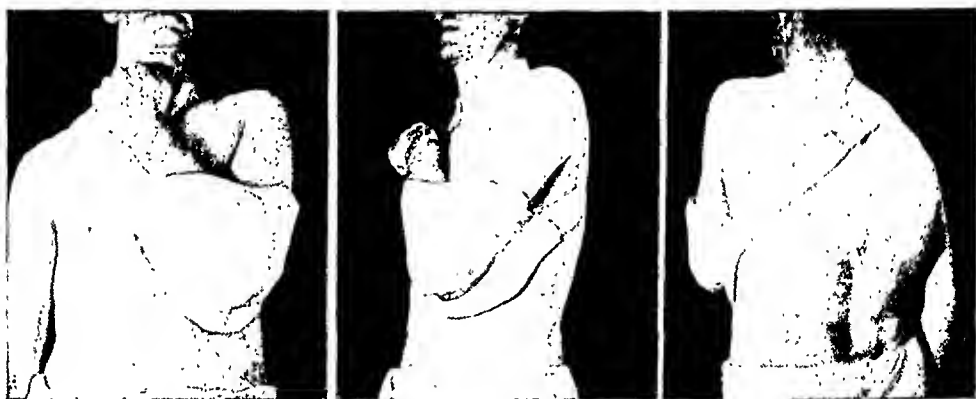


FIG. 213-215.—Apparatus for the treatment of fracture of the clavicle. Note the weight-bearing slab on the sound shoulder, which carries the weight of the elbow slab and arm of the affected side. The affected shoulder is held higher than normal. The elbow is fixed. The weight of the limb carries the shoulder backwards and outwards along an arc of a circle drawn with the elbow as its centre. The shoulder slab is made in the upright, the elbow slab in the recumbent, position.

as do most appliances used to treat fracture of the clavicle. The shoulder slab, in addition to providing a very comfortable bearing surface, makes rotation of the apparatus round the trunk practically impossible. This might occur if the lower slab were supported by a fabric band only, and in addition the latter would tend to ent into the skin and cause pain. Should the slabs cause trouble over the clavicle or bony points at the elbow, the plaster should be scraped away as described above. When it is necessary to remove the apparatus the patient should lie supine so as not to disturb the relationship of the fragments. The elbow- and shoulder-joints may then be exercised. The hand and wrist movements are not hampered in any way by this apparatus.

**Apparatus for Use in the Treatment of Fracture of the Shaft of the Humerus (Figs. 216-218).**—Note: The brachial portion of this apparatus is a modification of a splint originally designed by Mr. Fay Maclure, of Melbourne.

The position of rest for the upper limb is that shown in *Fig. 216*. When

a normal limb is slung in this way the elbow is well away from the side. Obviously it should be held in a similar position when the humerus is fractured. The elbow is bent at about a right angle and the wrist is supported by a sling or bandage from the neck. If a patient with a fracture of the shaft of the humerus is given morphia and left sitting for half an hour with his arm so supported, almost always muscular spasm disappears and the limb

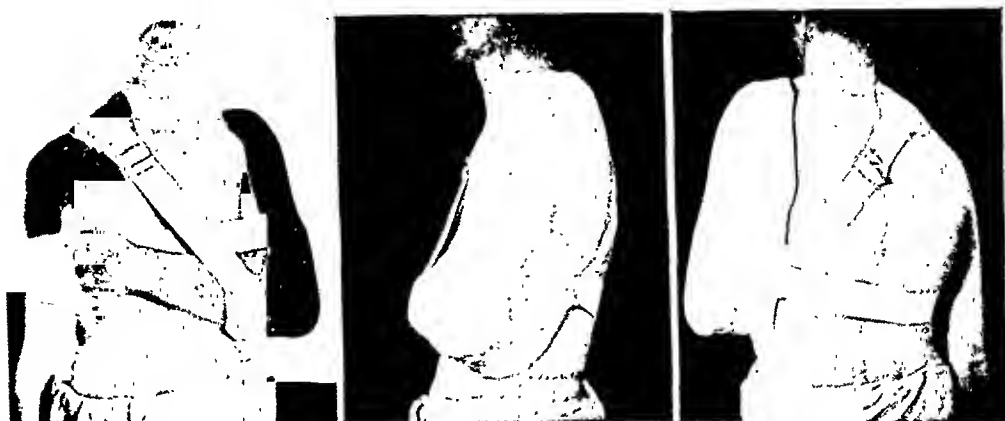


FIG. 216-218.—Apparatus for the treatment of fracture of the humerus. Note the weight-bearing slab and the brachial splint which consists of a postero-lateral gutter supported by a flange or buttress fitting on to the thorax. Both plasters are made on the patient in the sitting position, the wrist being slung from the neck in the position shown. The sling is omitted for the sake of clarity, but is of course worn with the apparatus. The elbow is well away from the side. The webbing bands pass right through the slabs.

takes on a normal appearance. It then remains to build a splint which will maintain the position. The splint used is a postero-lateral gutter supported above, posteriorly, and below by a buttress or flange fitting on to the thoracic wall. It is held in position by two webbing bands, one passing round the thorax, the other linking up with a weight-carrying slab similar to that used in the treatment of fracture of the clavicle. Both slabs are made on the patient in the sitting position. There is no constriction of the limb at any point. Immobilization is excellent. Jolting and jarring fail to move the apparatus relatively to the patient, and direct force applied to the slab is transmitted to the trunk without disturbing the injured segment of the limb. Inspection of the limb is easy. To remove the apparatus it is necessary to undo two buckles only. This should only be done whilst the patient is upright with the wrist supported by a sling from the neck.

**Injuries in the Region of the Wrist (Fig. 219).**—The arm and hand are supported on a table in the desired position, and a slab of the requisite length



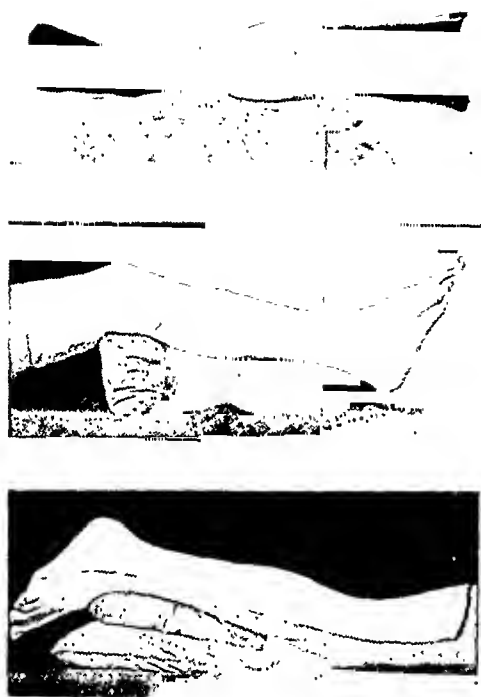
FIG. 219.—Splint moulded to support a wrist in flexion. A cock-up or any other form could be easily constructed.

is moulded on the upper aspect. In making very small slabs a gauze bandage is sometimes used instead of strips of scrim. Well-made slabs are more com-

fortable and efficient than wooden or metal splints in the treatment of injuries and diseases about the wrist-joint. The dimensions and shape of the slab and the attitude of the limb to be treated vary with circumstances. As with other splints of this type, removal is easy and re-application practically fool-proof.

**Splints for the Lower Limbs** (*Figs. 220-224*).—Plaster slabs are particularly useful in the treatment of fractures of the Pott's type. They may be moulded on any aspect of the limb. A back splint is made with the patient lying prone and the foot projecting over the end of the couch or large sand-bag. Side splints are very efficient, and the author frequently uses these, more particularly when inversion of the foot is desired or when there is a wound on the medial or lateral aspect of the limb. The patient lies on one or other side with the limb suitably supported on sand-bags. When both bones of the leg are fractured it may be necessary to control the fracture for a week or so by some method of extension prior to the application of a slab. Recently

Mr. E. T. Cato has brought to the author's notice a useful device. With the



**FIGS. 220-222.**—A posterior splint for the leg. *Fig. 222* pictures the position in which the limb is supported during construction of the plaster slab, but the support shown would be inadequate in practice. *Fig. 221* gives another view, and *Fig. 220* shows the splint alone. Note the accurate fit, the thin edges, the flexion at the knee. This splint is very strong on account of its shape, particularly where the foot and leg pieces merge. It is 'semi-cylindrical' throughout its length.



**FIGS. 223, 224.**—A lateral splint for the leg. *Fig. 223* shows the position in which the limb is supported during construction of the apparatus. The support shown is, of course, inadequate. In *Fig. 224* the splint is seen alone. Again the semi-cylindrical form is preserved.

patient lying prone traction is exerted on the foot through the medium of a bandage passed round the foot and ankle. A posterior slab is laid on as usual, extending well above the knee, which is slightly flexed. When set, the protruding ends of the bandage are cut away. There appears to be very little tendency for overlapping of the fragments to recur when this method is used.

### SUMMARY.

It is pointed out that many of the splints used to treat fractures are not well fitted for the purpose. A plea is made for the recognition of the many advantages attending the use of snugly-fitting plaster-of-Paris slabs. Whilst these are very efficient and comfortable for the patient to wear they also save the surgeon much time and trouble. The author's methods are briefly described.

The splints are made of plaster-of-Paris reinforced by strips of 'eight-ounce scrim', or hessian, and bound by a layer of gauze, and are moulded directly on the skin surface of the patient. Thus neatly shaped, thin-edged, light, strong, and accurately-fitting supports free from clumsy wrinkles are obtained, which are very easily managed. Wherever possible the splints approach in section the semi-cylindrical shape, which makes for strength and efficient support whilst avoiding any danger of constriction of the part.

Careful placing of the injured part in the best possible position is of first importance, and this is done deliberately before commencing operations. The part is firmly supported on sand-bags, and during subsequent manœuvres is not disturbed in any way. In certain instances webbing bands provided with buckles are built into the splints and bandages are dispensed with. Cotton-wool is used only on the limb surface which is *not* covered by the plaster, to permit of swelling of traumatized tissues when bandages are used. A single layer of very thin material such as silk or gauze may be worn between skin and plaster.

Methods of dealing with various difficulties are discussed. Certain individual splints are described, but it is pointed out that these are only examples of what may be done with the method. The surgeon who masters the technique and has sufficient ingenuity will be able to devise for himself splints of infinite variety. If he takes the simple precaution of having the materials always at hand, he will be prepared to produce an efficient apparatus at any time. Success depends upon attention to detail, and practice. The more the surgeon uses the method, the more useful will he find it.

It is not intended to imply that there is never any need for other forms of apparatus, nor that the splints are more than an aid to the surgeon in the treatment of fractures, arthritis, and so on.

The author wishes to convey his thanks to numerous colleagues from whom he has received useful hints, to patients who have taught him much, to many house surgeons who have cheerfully helped him in the production of photographs, and lastly to Mr. Fay Maclure who first brought the idea of plaster slabs before him and who has since then never failed to take a kindly and helpful interest in the development of the present methods.

## REGENERATION OF THE INTERNAL SEMILUNAR CARTILAGE AFTER OPERATION.

By A. GIBSON, WINNIPEG.

IN an excellent monograph on internal derangement of the knee-joint<sup>1</sup> there is related the history of a case in which both semilunar cartilages were removed 'elsewhere', the internal on Aug. 8, 1921, the external on April 5, 1922, yet "on full exposure it was seen that both semilunar cartilages were present. The internal was normal. . . . The fact that this patient had undergone operations by the 'classical' routes upon both cartilages and a more complete exposure showed them both to be present is particularly instructive. The finding of a cartilage alleged to have been removed has probably befallen most of us, and this fact is a strong argument in favour of a complete exposure." Not necessarily!

On Oct. 8, 1928, J. W. O., machinist, came under my care. On May 30, 1928, he was pulling down a big wrench with all his weight. His right knee came violently against a plate under his bench. From that time onward he complained of something slipping in the right knee-joint. On Aug. 10, 1928, he was operated on 'elsewhere' and had not been back at work since. On the morning of Oct. 8 the joint seemed to lock.

On examination an oblique sear was present on the medial side of the right knee. There was considerable swelling of soft parts and considerable heat. Extension was possible to an angle of about 165°. Flexion was possible to about 90°. Examination by means of the X rays showed no loose body in the joint. There was atrophy of the patella and of the adjoining part of the femur.

On Oct. 23, 1928, a 'complete exposure' was made by parapatellar incision. The internal semilunar cartilage was in position; its posterior extremity was much thickened. The synovial membrane of the joint was hyperæmic. A considerable part of the internal semilunar cartilage was removed and was sent for microscopic examination. The report was as follows: "Specimen consists of dense fibrous tissue only—no cartilage cells." *Fig. 225* shows a normal human meniscus, and *Fig. 226* that of this patient. With this finding in mind it was decided to submit the matter to experiment.

On May 8, 1929, two dogs were operated on; the knee-joint was opened and part of the internal semilunar cartilage removed by the 'classical' route. The dogs were turned loose on a farm for the summer and showed no disability. On Oct. 4, 1929, one of them was brought in and sacrificed, and both knee-joints were examined. There was apparently complete restitution of the internal semilunar over the area of operation (*Figs. 227, 228*). Microscopic

examination showed the specimen to consist of dense fibrous tissue only with no cartilage cells.\*

Mandl<sup>2</sup> has described three cases in which a 'delicate structure' replaced

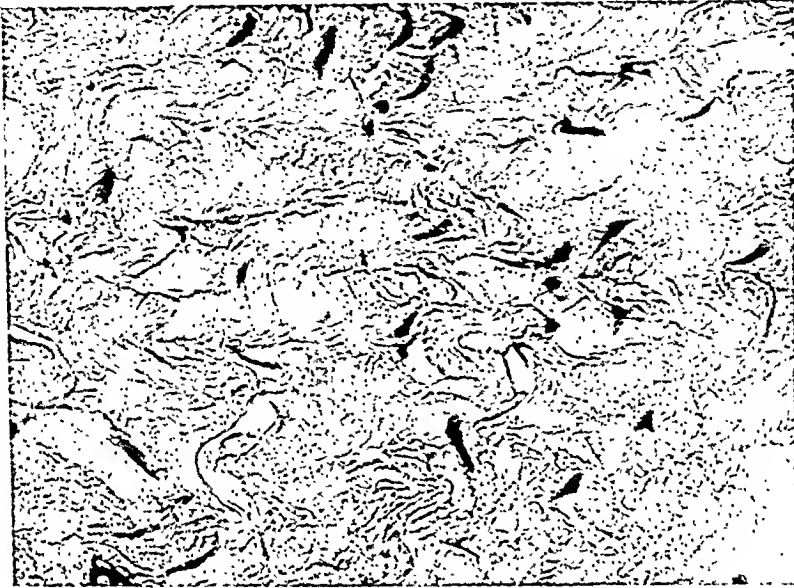


FIG. 225.—Normal human meniscus. ( $\times 400$ .)

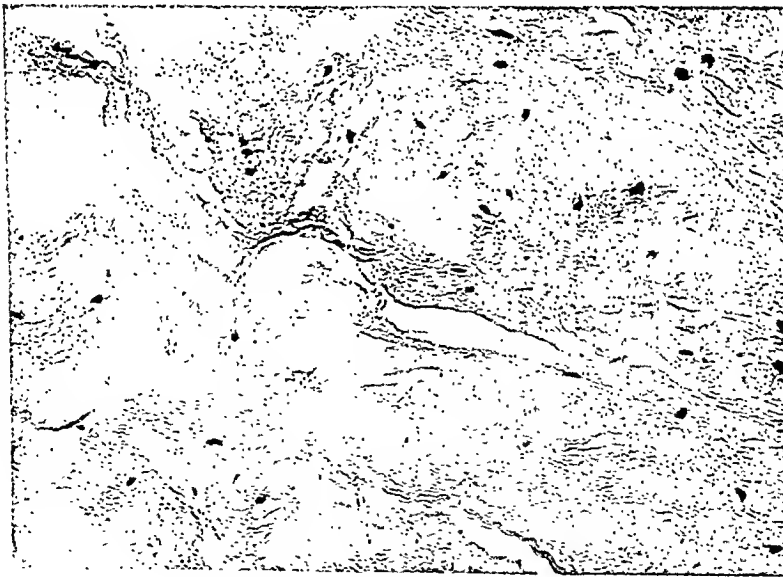


FIG. 226.—Meniscus of J. W. O. ( $\times 250$ .)

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\* This specimen was shown at the Orthopaedic Section, British Medical Association, Winnipeg, in August, 1930.

the excised semilunar cartilage. In the patient and the dog mentioned above, the replacement was in no way delicate. It was if anything superior to the original.



FIG. 227.—Dog. Normal meniscus. ( $\times 400$ .)

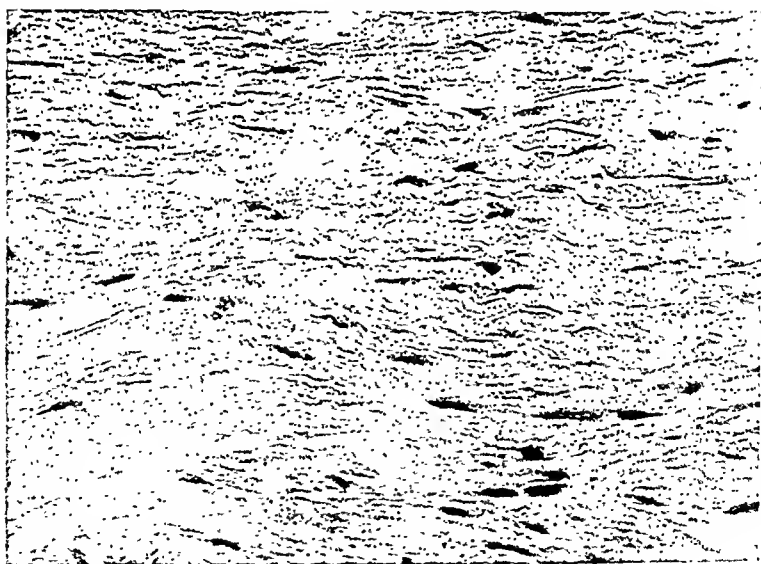


FIG. 228.—Dog. Regenerated meniscus. ( $\times 400$ .)

Lukjanov and Pokrovski<sup>3</sup> operated on 37 dogs, of which 12 died post-operatively (!). They found that "removal of the menisci, partial or complete, has resulted in regeneration of the cartilage. These differ from the normal

ones neither in form nor consistency; they are only less glossy and movable. The newly formed semilunar cartilages are functionally and statically of equal value with the ones removed."

It would seem, therefore, that we have consistently underestimated the restorative power of the body in assuming that a semilunar cartilage when excised is excised for good. Our patients generally ask us what degree of function they may expect after the operation of meniscectomy, to which we generally reply with confidence that full function may be looked for. The experimental findings mentioned above may furnish an anatomical as well as an empirical basis for the faith that is in us.

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- <sup>1</sup> FISHER, A. G. TIMBRELL, *Internal Derangement of the Knee-joint*, 70.
- <sup>2</sup> MANDL, F., *Zentralb. f. Chir.*, 1929, 3265 (abst. in *Surg. Gynecol. and Obst.*, 1930, Aug. 147).
- <sup>3</sup> LUKJANOV and POKROVSKI, *Jour. Sovremennoi Chir.*, 1929, iv., 946 (abst. in *Jour. Bone and Joint Surg.*, 1930, Oct., 984).



## EXPOSURE OF THE KNEE-JOINT.

BY H. B. DEVINE, MELBOURNE.

THE indications for operation on the knee-joint may be divided for practical purposes into two classes, according to whether a definite diagnosis may or may not be made :—

1. First, those conditions where the lesion is localized and where it is possible to make a definite diagnosis, and the surgeon is reasonably certain that if he makes an exposure over the affected part of the joint, he will be able to deal with the lesion. Such, for instance, is internal derangement of either of the menisci. Here, if the classical syndrome is present, there is very little possibility of a mistake in diagnosis and that an exploration will be unnecessary. The best surgery in this case is to disturb the main part of the joint as little as possible, for we know that the knee-joint has a small margin of error, both as regards operative trauma and sepsis.

2. The second class includes all those conditions of the joint which give a syndrome like an internal derangement of a meniscus and which are mistaken for it—for example, loose bodies or nipped hypertrophic synovial fringes. In these patients a normal cartilage is often removed in the belief that it is diseased, because the surgeon is satisfied, on account of the inadequate exploration possible, that there is no other lesion. Similarly, if a loose body or a nipped synovial fringe is found, it is impossible to make sure that there are not others. A small injury to the crucial ligaments sometimes cannot be recognized through the limited exposure. For these reasons, and others that could be mentioned, the symptoms and disability of the patient recur.

It is surgically more correct in these cases to treat the joint as one would the abdomen in indefinite abdominal conditions—that is, employ an operation which approaches in a simple way the middle of the joint, which brings the whole joint under vision without an unnecessarily big opening, which can be closed without using non-absorbable material, and which heals quickly enough to allow early movement.

There are three recognized methods of exposing the knee-joint : Kocher's operation,<sup>1</sup> Timbrell Fisher's,<sup>2</sup> and the operation (after Hamilton Russell<sup>3</sup> and Sir Robert Jones<sup>4</sup>) in which the patellar tendon, the patella, and quadriceps tendon are split longitudinally. In all these operations the joint is approached through longitudinal incisions, which must divide joint structures extensively in order to give the necessary exposure of the transverse joint surface of the tibia. Anyone who has seen the beautiful exposure of the joint that is obtained when operating on a fractured patella will appreciate that the natural approach to the knee-joint should be through a transverse incision.

The operation described below is based on an approach through a transverse incision by means of which the knee-joint may be explored and operated on in a manner comparable with explorations of the abdominal cavity—that is, by its means, perfect exposure, complete exploration, certain control of bleeding, and physiological closure may be obtained.

### TECHNIQUE OF THE OPERATION.

The steps of the operation are as follows:—

1. A curved incision, the middle of which is about half an inch below the lower margin of the patella, is made (*Fig. 229*). A flap of skin and fat is turned up and protected in the usual way.

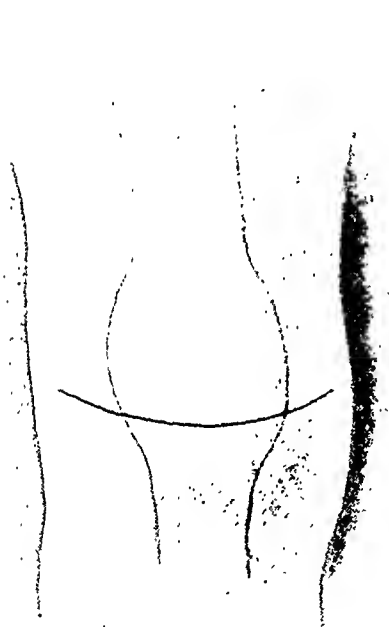


FIG. 229.—Incision for exposure of the knee-joint.

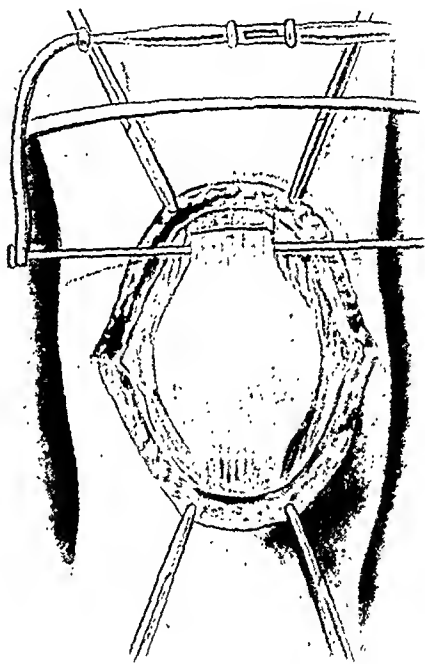


FIG. 230.—Beginning of the longitudinal (coronal) saw-cut through the patella.

2. With a butcher's saw, starting in the quadriceps tendon about half an inch above the upper margin of the patella (*Fig. 230*), a cut is made in a direction which slopes backwards through the tendon of the quadriceps, the patella, and the patellar ligament, coming out of the latter on the posterior surface half an inch below the lower margin of the patella. The object is to separate the anterior half of the patella from the posterior portion without disturbing the cartilaginous surface, and to commence and complete the saw-cut so as to leave attached to each plane of the patella a strong piece of tendon by means of which the patellar segments subsequently may be united firmly with catgut sutures.

3. The assistant bends the knee; the patellar fragments separate and the lateral expansions of the joint stretch tightly. The surgeon now puts a small hook under the lower margin of the upper fragment and turns its cartilaginous surface over (*Fig. 231*), and as the knee continues to bend, he applies his knife in the direction of the vessels to the tightly stretched lateral expansions till he has exposed the joint sufficiently for his purpose. As a rule there is little bleeding.

4. Manipulations may now be carried out with very little instrumentation and certainly without any handling, because the joint is so accessible. All bleeding is stopped and the joint is left quite dry.

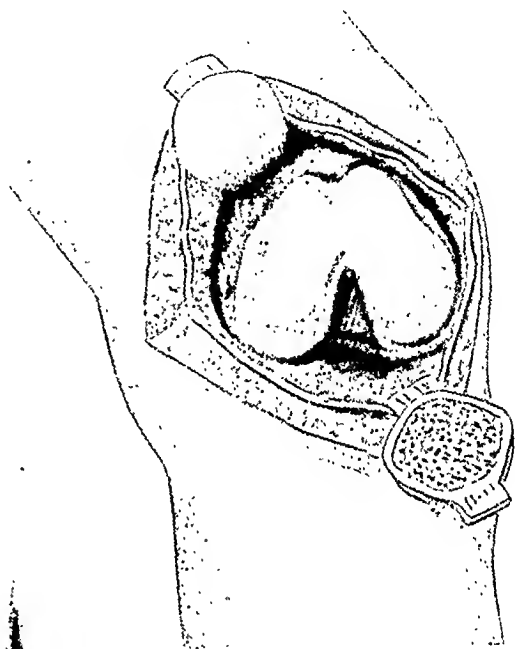


FIG. 231.—Full exposure of the knee-joint.

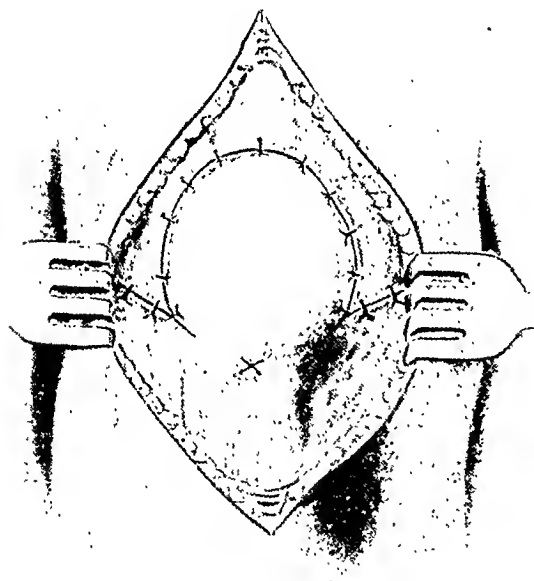


FIG. 232.—The patella sutured back into position.

5. The assistant now extends the leg, when it will be found that the opening in the joint closes and the sliced patellar fragments lie nicely in apposition. The fragments are then fixed together by suture, with catgut, of the obliquely sliced patella and quadriceps tendon. The synovial membrane and the quadriceps expansion are closed separately (*Fig. 232*).

Since it is not possible to guard against the occasional unexpected reactionary hæmorrhage, which has dangerous potentialities as a cause of post-operative complications, the use of a tourniquet in knee-joint operations should be regarded as dangerous. In the operation described there is no

need for a tourniquet, for not only is bleeding avoided by operating through a comparatively bloodless area, but since it is possible to see what is happening, bleeding may be easily and certainly controlled by ordinary methods.

The large patellar raw surfaces make for a firm and early union, and in ten days it is difficult, with an X-ray, to see the line of contact (*Fig. 233*).

The exposure makes the joint so accessible that manipulations can be carried out with the greatest precision and gentleness.

It is our experience that it is an operation which leaves nothing to chance; and that, if the surgeon utilizes his skill and cunning, he can leave a joint which heals rapidly with little reaction and no adhesions, and allows early function.

We could quote many instances where in doubtful cases of internal derangement we were tempted to do the limited operation, where we should have hesitated to do the more formidable recognized exploratory operations, and where the operation described disclosed unexpected conditions such as a hidden loose body, a strong string-like adhesion associated with the crucial ligaments, unsuspected early tubercle, partial injuries to the attachments of the crucial ligaments, osteo-arthritic manifestations, and other lesions.



FIG. 233.—Skiagram showing union of patellar segments ten days after operation.

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- <sup>2</sup> FISHER, A. G. TIMBRELL, *Internal Derangements of the Knee-joint, their Pathology and Treatment by Modern Methods*, 1924. London.
- <sup>3</sup> RUSSELL, R. HAMILTON, "On Access to the Knee-joint by Anterior Section of the Patella", *Interecol. Med. Jour. of Aust.*, 1906, ix, Aug. 30, 441.
- <sup>4</sup> JONES, SIR ROBERT, *Injuries of Joints*, 1915. London.

**CHOLECYSTITIS GLANDULARIS PROLIFERANS (CYSTICA).**

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CHOLECYSTITIS glandularis is a circumscribed or diffuse thickening of the wall of a chronically inflamed gall-bladder, characterized by excessive proliferation of and invasion of the wall by epithelium, forming crypts, with dilatation of some of these crypts to form cysts.

This condition has been described many times under several names: adenoma,<sup>8, 30</sup> polyp,<sup>28</sup> cystadenoma,<sup>15</sup> adenomatous polyp,<sup>31</sup> papilloma,<sup>9, 21</sup> fibro-adenoma,<sup>10</sup> fibromyoma,<sup>32</sup> carcinoma,<sup>27</sup> cholesterol cyst,<sup>8, 13, 24</sup> preeancerous proliferation,<sup>33</sup> and sometimes has been discussed without being regarded as of any special significance.<sup>12</sup>

The routine examination of 400 gall-bladders removed at operation for cholecystitis or cholelithiasis demonstrated 38 organs (9.5 per cent) showing this type of change. Twenty-five well-marked specimens were chosen to form the basis of this paper. Since they possessed certain characteristics in common, which distinguished them from other conditions, they were grouped together.

**Terminology.**—A study of the various conditions which resemble those referred to here and which are described in the literature shows that they all occur in association with chronic cholecystitis and manifest morphological similarities.

In 1922 Bodnár<sup>5</sup> described a diffuse form—the most characteristic feature being the presence of cysts of various sizes in the wall of the gall-bladder, and he styled the condition ‘cholecystitis cystica’ in analogy with cystitis cystica and ureteritis cystica. Although this is an excellent term and might be used to apply to many of the examples, it does not embrace the cases where cystic change is not predominant.

The term ‘cholecystitis epithelialis heterotopica’ suggested itself, but this covers too many cases, since a large number of chronically inflamed gall-bladders show heterotopic proliferation of epithelium but not the characteristic macroscopic appearances. This term, also, is too unwieldy for ordinary use. We would suggest that a better name is ‘cholecystitis glandularis proliferans’. Though an etymological hybrid might be considered undesirable, the term is suitable from every point of view except that of the classicist, but we have innumerable examples in medical literature as precedents for such a transgression.

**GENERAL CHARACTERISTICS.**

As stated, cholecystitis glandularis presents itself as a thickening in some portion or portions of the wall of the gall-bladder, this thickening being

honeycombed by epithelium-lined duets. It may occur in any part, but we have observed it most frequently and characteristically in the fundus of the organ. In the cases examined there has been evidence of chronic cholecystitis in the wall away from as well as in the specially thickened tissue. This special tissue sometimes is demarcated abruptly from the surrounding gall-bladder wall, but in other cases it merges gradually into the neighbouring parts.

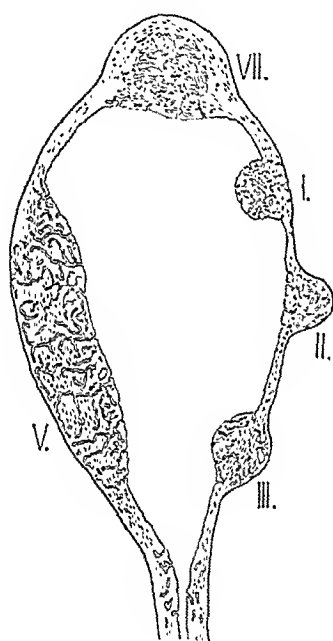


FIG. 234.—Diagram illustrating some of the forms of cholecystitis glandularis which have been encountered. For fuller details see Fig. 235.

Rarely the tissue may have an apparent capsule. Macroscopic cysts are not an invariable feature of the group under consideration.

**The Macroscopic Appearance.**—Several appearances, all merging from one to another, may be observed.

The thickening of the wall may project into the cavity of the gall-bladder (Figs. 234 I, 235 I). This

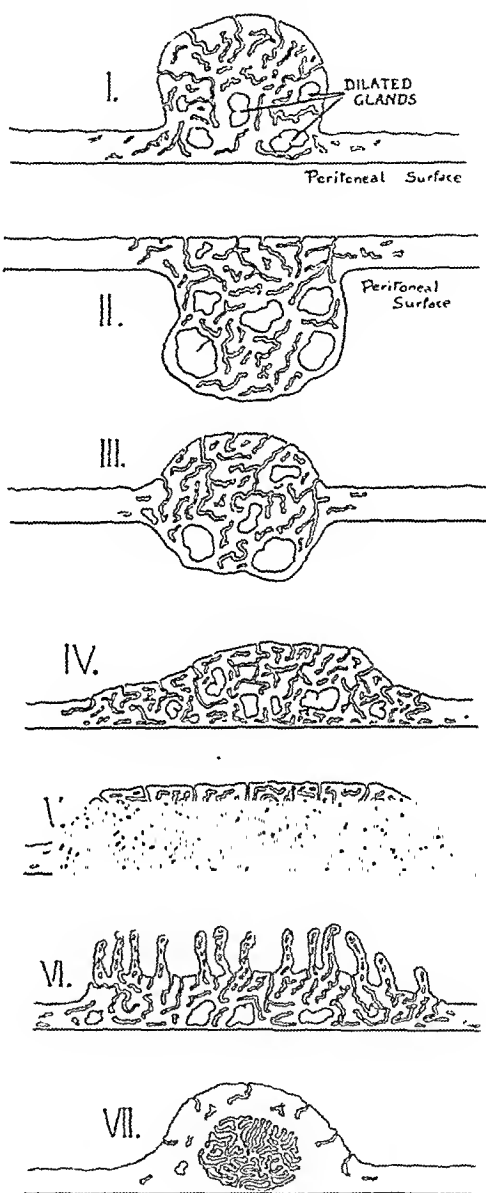


FIG. 235.—Diagram illustrating the various types of cholecystitis glandularis which have been encountered. I, Localized nodule projecting from the mucous surface; II, Localized nodule projecting from the peritoneal surface; III, Localized thickening of the wall causing a projection from both surfaces; IV, Intermediate form between the localized and diffuse types; V, Diffuse form; VI, Variety with papillary developments on the mucous surface; VII, Localized nodule in the wall, apparently unconnected with the surface (adenoma).

projection is usually sessile, but we have observed one example in which the base had a smaller diameter than the polyp, i.e., it was slightly pedunculated. The mass may become definitely pedunculated in a manner similar to the polypoid projections in the intestine. On gross section the tissue may be reddish, brown, greyish-white, or yellow in colour, and shows a number of open spaces.

The projection may take place into the peritoneal cavity (*Figs. 234 II, 235 II*). This is usually small and sessile. In one example the diameter of the base of attachment was equal to the diameter of the nodule. The peritoneum which covers the nodule is intact. The mucous membrane may be ulcerated, but is usually intact.

The thickening of the wall sometimes enlarges from the mucous as well as the peritoneal surface. This presents no special features, but combines those of the forms just described (*Figs. 234 III, 235 III*).

These localized conditions merge into those in which a considerable portion of the wall is involved (*Fig. 235 IV*). Since the thickening is usually uniform, no projection is seen from the peritoneal surface, and the thickening appears to take place towards the lumen.

The generalized forms of cholecystitis cystica (*Figs. 234, 235 V*) may involve a considerable portion of the wall of the gall-bladder. We have observed, in one case, as much as three-quarters of the wall involved by this change. Rarely a papillary development into the cavity of the organ accompanies the changes in the wall, and may be recognized by a characteristic shaggy appearance when the projections are long, and a velvety form when they are short. This variety is the one which is most frequently regarded as carcinoma (*Fig. 235 VI*).

Occasionally a nodule of tissue is seen which does not possess an obvious connection with the lining epithelium (*Figs. 234 VII, 235 VII, 236, 237*). These cases, when not diagnosed as carcinoma, are referred to as adenoma. They are described as arising from 'aberrant gland tissue'. The undesirability and fallacy of such opinion will be discussed.

The cysts which occur in these tissues are usually small, though readily apparent, but sometimes they become large and constitute the major portion of the lesion. They contain fluid laden with cholesterol and, rarely, cholesterol concretions.<sup>13, 24</sup>



FIG. 236.—Specimen showing a localized area of cholecystitis glandularis at the fundus of the gall-bladder.

**Microscopic Changes.**—On microscopic examination a large number of closely intermingled changes are encountered. The most obvious is the

**EPITHELIAL PROLIFERATION.**—This activity of the epithelium is shown in two ways: (1) By downgrowth of the epithelium into the subjacent tissues; (2) By proliferation *in situ*, with multiplication of the layers of cells.

1. The downgrowth of the epithelium into the subjacent layers is the essential feature of the condition of cholecystitis glandularis. The glandular crypts retain the typical columnar epithelium with a definite basement membrane. There is no evidence of neoplasia; all suggestion of disordered growth and invasion with destruction of the surrounding tissue is absent.

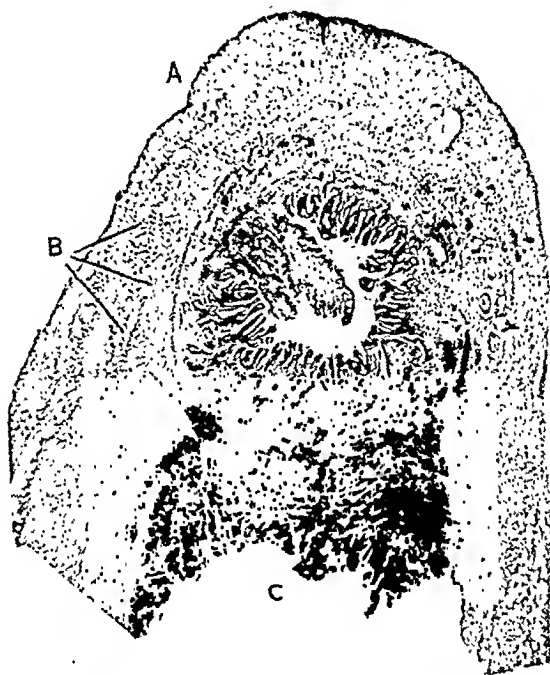


FIG. 237.—Section of the nodule in the fundus of the gallbladder. The connection of the epithelium of this nodule with that of the surface is shown by serial sections. A, Peritoneal surface; B, Fat; C, Connection of the epithelium of the nodule with that of the surface.

in Fig. 236.  
was shown  
( $\times 3$ .)

The glandular crypts pass through the muscular coat and finally reach the subperitoneal layer. The course followed by these crypts is often very tortuous, and these tortuous portions cut in section give the appearance of numerous 'glands' (Fig. 244). The tortuosity of these crypts in the deeper layers is of paramount importance, since it may be assumed readily that there is no connection between the 'glands' observed in the deeper layers and the surface epithelium.

The cystic spaces, which are found particularly in the submucous and subperitoneal coats, are lined by a single layer of columnar cells, but in the larger cysts these become cuboidal or even flattened. Adjacent cysts may coalesce to form larger cysts (Fig. 238).



2. Local proliferation of the epithelium results in the formation of two or three layers. These are quite regular in their formation, however, both with regard to size, shape, and position, only a few mitotic figures being present and the basement membrane intact. Sometimes they approximate in appearance to squamous epithelium.<sup>14</sup>



FIG. 238.—Section of a localized nodule which projected from the peritoneal surface of the gall-bladder. A, Mucous membrane; B, Peritoneum. ( $\times 6$ .)

These changes have been regarded as evidence of carcinomatous or, at least, pre-cancerous development. There is, at present, no evidence for this assumption.

**METAPLASIA.**—The alterations of the epithelium from the typical gall-bladder type to varieties resembling cells typical of other portions of the bowel make the examination of the sections of cholecystitis glandularis a fascinating study. The types of epithelium found are: (1) Columnar epithelium with or without goblet cells—an epithelium of intestinal type; (2) Mucous glands; (3) 'Gastric' glands. These structures show the alteration of gall-bladder epithelium to various other forms of alimentary canal epithelium.

1. *Columnar Epithelium.*—In many of the epithelium-lined crypts the epithelium is of a tall columnar character, with basal nuclei (Fig. 241). The regularity of the size and position of these nuclei is a characteristic feature of many of the sections.

*Goblet Cells.*—The cells of the epithelium of the gall-bladder differ from those of the intestine in that all the cells produce mucin at the same time, but only in the form of small droplets. Thus in the normal gall-bladder goblet cells are infrequent. In the inflamed organ, however, goblet cells occur in a large proportion of cases.<sup>14</sup> These are to be found in most of the examples of cholecystitis glandularis.

2. *Mucous Glands* occur not uncommonly in chronic cholecystitis, having been found 185 times in 250 consecutive cases described by the writer elsewhere.<sup>14</sup> They occur in the



FIG. 239.—Portion of the thickened wall of a gall-bladder, showing the epithelium-lined crypts and the associated glands. ( $\times 30$ .)

# CHOLECYSTITIS GLANDULARIS PROLIFERANS 315

majority of the examples of cholecystitis glandularis, and were described in 1905 by Aschoff.<sup>2</sup>

They resemble mucous glands occurring elsewhere. The epithelial cells are arranged in small alveoli, the nuclei are round or oval and are situated in the middle of the cell, the position varying with the activity of the cells, and the protoplasm stains deeply with mucicarmine (Figs. 240, 243). There is no doubt that these glands arise from the surface epithelium either directly or from the crypts, since on section direct continuity of the epithelium can easily be traced.

3. 'Gastric' Glands.—In a number of cases of chronic cholecystitis in which mucous glands occur, other glands resembling the glands of the

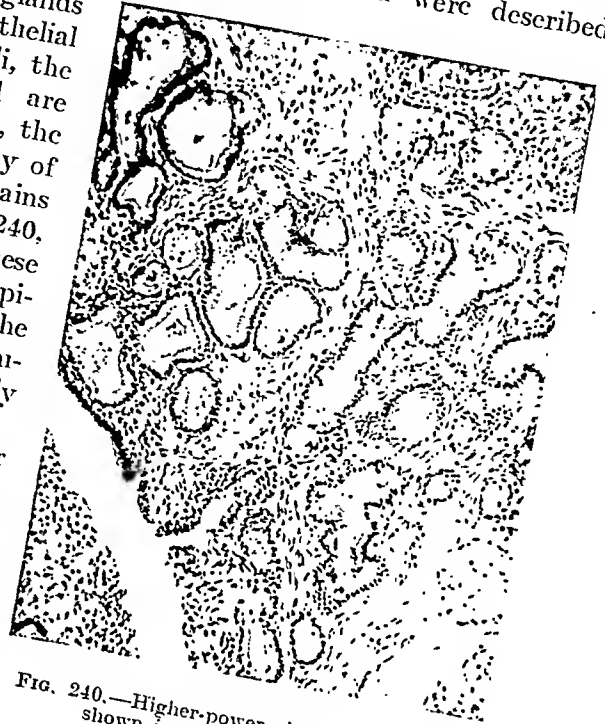


FIG. 240.—Higher-power view of the tissue shown in Fig. 239. ( $\times 100$ .)



FIG. 241.—Portion of an epithelium-lined down-growth showing the columnar cells resembling intestinal epithelium. The regularity of the size, shape, and position of the nuclei demonstrates the innocent nature of the epithelium. ( $\times 810$ .)

stomach and Brunner's glands of the duodenum are encountered. In structure they imitate mucous glands (Figs. 244, 249), but the alveoli contain clearer cells than those of the mucous glands, and the nuclei are crescentic in shape and situated in the basal portion of the cell. The protoplasm does not stain with mucicarmine. They are less common than the mucous glands, occurring in about 66 per cent of the cases in which these are found.<sup>14, 19</sup> They appear to arise usually by a modification of the mucous glands, but I have observed them in relationship with the gall-bladder epithelium without the intermediation of mucous glands.

RELATIONSHIP OF EPITHELIUM TO MUSCLE LAYER.—In order to reach the subperitoneal layer, the epithelial crypts penetrate the submucous and the muscular layers. The interruption of the muscular layer is a well-marked



FIG. 242.—Portion of tissue from an example of cholecystitis glandularis showing a 'lymphoid nodule'. ( $\times 110$ .)

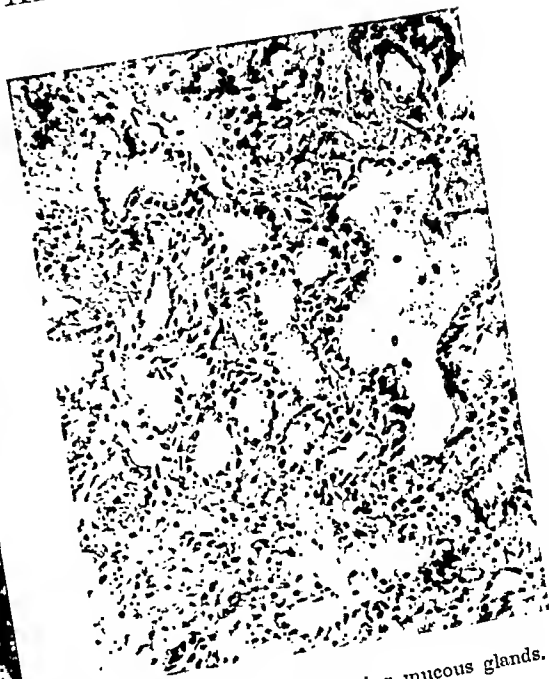


FIG. 243.—An area showing mucous glands. ( $\times 160$ .)

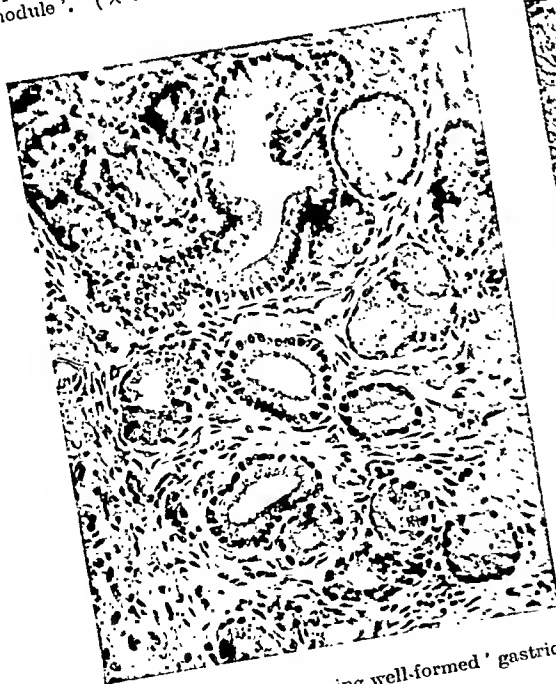


FIG. 244.—Section showing well-formed 'gastric' glands. ( $\times 160$ .)



FIG. 245.—Epithelium-lined space in the wall of a gall-bladder showing the close association of the muscle tissue to the cavity. ( $\times 40$ .)

feature of some sections (*see Fig. 238*). Frequently the muscle fibres are found to encircle, almost completely, groups of 'glands'.

There can be little doubt that in many of the examples of chronic inflammation of the gall-bladder there is proliferation of the smooth muscle. The amount of muscle in the wall of some of the thickened organs is much in excess of that found in the normal gall-bladder.

It is probable, therefore, that some of the muscle found around the epithelial groups is new formation; this opinion is expressed in the nomenclature used by some writers,<sup>3, 20</sup> 'adenomyoma', etc. It might be suggested that the arrangement of muscle around the glands is a response to function, i.e., new muscle is formed to give rise to a new small 'gall-bladder' in the wall of the organ (*Fig. 245*). On the other hand, the muscle lying around some of the larger groups probably arises from displacement of the existing muscle (*Fig. 246*).

In some examples the proliferation of the epithelium occurs for the most part on the mucous membrane aspect of the muscular layer, and in others it occurs largely on the peritoneal aspect. This relationship of the greater part of the proliferating epithelium to the muscular coat appears to be the circumstance deciding whether the nodule will project internally or outwards into the peritoneal cavity.

**CONNECTIVE - TISSUE CHANGES.**—The changes in the connective tissue (in chronic inflammation) may be considered in two groups: (1) Those apparently associated with the epithelial proliferation; (2) Independent changes.

1. Sometimes collections of round cells are found near the surface in association with epithelium which has become very similar to that of the bowel. These seem to correspond to the solitary nodules of the bowel (*Fig. 242*). This is, of course, hypothesis, but the association with the epithelium mentioned and the absence of other cells of chronic inflammation in the lymphoid collection are the reasons for the assumption.

Around the epithelial crypts the connective tissue may be of loose texture or definitely fibrous.



FIG. 246.—Portion of the nodule shown in *Fig. 236*. There is some atypical proliferation of the epithelium, but the glands are well differentiated. The arrangement of these glands suggests that they are coiled tubes cut in many places. This is proved by serial sections. ( $\times 85$ .)

2. The proliferation of connective tissue cells observed in all chronic inflammatory conditions is found here, and may be extreme. On several occasions we have investigated gall-bladders, which we were convinced were



FIG. 247.—Portion of the wall similar to that shown in *Fig. 246*. Coiled tubules, cut in section, are present, principally of the mucous type, but also a few of the 'gastric' variety.

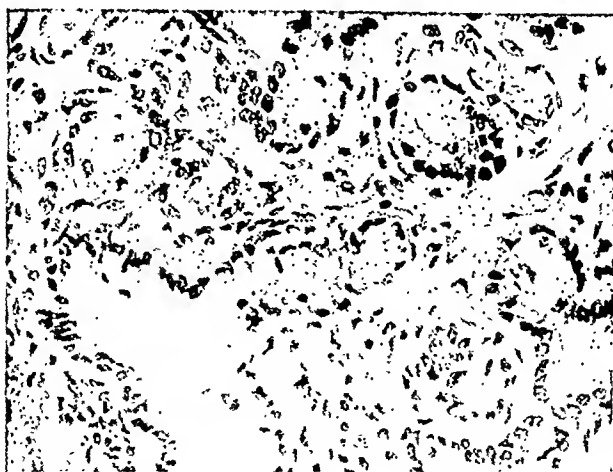


FIG. 248.—Photomicrograph showing the mode of formation of the glands as an outgrowth of a crypt. ( $\times 200$ .)

of the cholecystitis glandularis type, but found only extreme fibrosis. Some of these, however, may be related, since in some cases evidence of the previous presence of glands is to be found in the occurrence of small spaces

sometimes still containing a few epithelial cells or of degenerating epithelial cells lying in the connective tissue.

The excess development of fat is striking in some organs, either as fat purely, or with a mixture of connective tissue. This tissue sometimes merges into lymphoid tissue, which, however, is distinct from the lymphoid nodules referred to above.

Any of these changes which occur in ordinary cases of chronic cholecystitis may be found in the condition described here.

**Acute Inflammation.**—When acute inflammation supervenes on chronic inflammation, the characteristic phenomena appear in the subepithelial tissues, and, depending on the degree of the stimulus and the time at which it was examined, hyperactivity and then destruction of the epithelium may be found.

The inflammation spreads down the crypts, and similar changes occur here. The destruction of the deeper epithelium may be an explanation of not finding this in cases where it was expected. In two cases of small abscesses in the wall of the gall-bladder, a few large epithelial cells were found, suggesting that the site

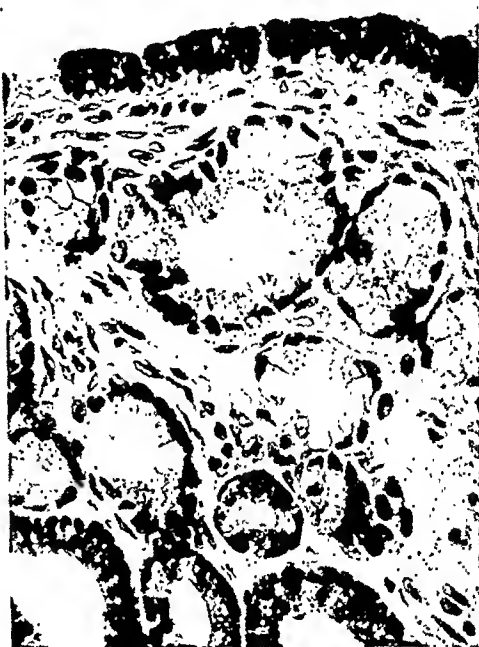


FIG. 249.—Higher-power view showing the structure of the 'gastric' glands. ( $\times 360$ .)

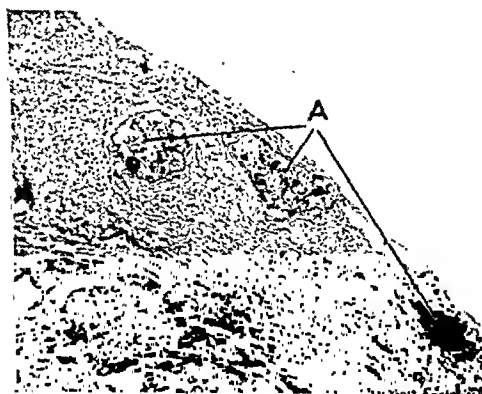


FIG. 250.—Small subperitoneal cysts containing cholesterol and bile pigment. ( $\times 30$ .)

of the abscess was originally an epithelium-lined space.

The relationship of the crypts to the peritoneum probably affords an explanation of the observation that some patients with acute cholecystitis have a peritonitis early, whereas others do not do so—excluding for the moment other factors, such as the virulence of the organism, etc.

**Other Complications.**—Obstruction of the cystic duct by a pedunculated 'adenoma' is described by the Mayos in Keen's *Surgery*. A calculus in a cyst in the wall of the gall-bladder may be mistaken for a calculus in the cavity of the gall-bladder.

**Relationship to Malignancy.**—This problem, as in all parts of the body, is extremely difficult, and apparently convincing arguments may be offered for diametrically opposed opinions.

It seems to us reasonable and probable that neoplastic development could arise in the epithelium of cholecystitis glandularis. On the other hand, we have not observed, in the few examples that have come to our notice, any instance of changes which would withstand critical examination by the usual criteria for malignancy. The regular arrangement of the cells, the

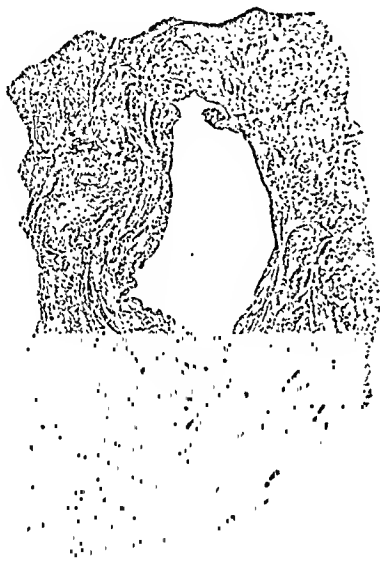


FIG. 251.—Portion of a wall of a gall-bladder showing cystic spaces which are not lined by epithelium. The organ had been the site of both chronic and acute inflammation, and the cysts were probably lined previously by epithelium which had been destroyed. (See Fig. 252.) ( $\times 11$ .)



FIG. 252.—Higher-power view showing the nature of the lining of the cysts seen in Fig. 251. The greater part of the cavity of the gall-bladder had a similar lining. ( $\times 75$ .)

normal polarity and the regular character of the nuclei, the presence of basement membrane, the degree of differentiation of the metaplastic glands all militate against a diagnosis of malignancy.

### CLINICAL CHARACTERISTICS.

Cholecystitis glandularis presents no special clinical features. It has been observed, in this series, in patients whose ages ranged from 38 to 72. All the patients were females.

In every case the gall-bladder was removed on account of signs and symptoms of chronic cholecystitis; in 9 of them acute inflammation had supervened.

Gall-stones were present in 21 out of the 25 cases. In the 4 other cases, record of the presence or otherwise of stones was absent. Its greatest importance rests in its possible confusion with malignant disease. A thickening of the wall, particularly when considerable fibrosis rendered this very hard, and nodular projection several times simulated and

led, at operation, to the provisional diagnosis of malignancy. Even microscopically two cases were returned as carcinoma. The subsequent history shows that the patients have had no ill effects after a period of seven years, even though in one case the thickening in the wall, examined microscopically, extended for 2 in. along that aspect of the wall which was in contact with the liver.

As stated, the condition was found in 9.5 per cent of 400 gall-bladders examined, but this result cannot be accepted as an indication of the frequency of occurrence, since the cases were, unintentionally, picked. For example, in addition to material derived from hospital and our own practice, a number of the specimens were sent to the writers by colleagues (to whom they would express their grateful appreciation) who knew of the interest evinced by us in the subject, and other 'less interesting' material was not examined.

In comparison with this, there are the figures of 'innocent tumours' observed by other writers :—

Mayo	-	-	107 examples of papilloma in	2538 gall-bladders	(4.21 per cent)
Irwin and McCarty <sup>11</sup>	85	„	„	2168	(3.92 „ )
Abell <sup>1</sup>	8	„	„ tumours in	288	(2.78 „ )

The difference between these and the figures of this paper is due in part to the 'picking' of cases described here, and also to the inclusion of examples showing a degree of development insufficient to give rise to a diagnosis of neoplasm.

## DISCUSSION.

The importance of grouping the various forms of this condition together is shown by the many opinions—as expressed by the terminology—promulgated regarding its nature. The hypotheses put forward are that it is : (1) Heterotopic tissue displaced in foetal life, i.e., a cell rest. (2) Neoplastic development—(a) malignant, (b) innocent. (3) A precancerous change.

1. **Heterotopic Tissue Displaced in Foetal Life.**—The brilliant hypothesis of Cohnheim that masses of aberrant tissue resulted from the segregation of cells during foetal life and that tumours could arise from these has gained a firm hold on pathological opinion. However, careful examination of structures apparently conforming with this suggestion demonstrates that all gradations may be found from the normal through minor alterations up to the fully developed lesion. This is shown particularly well in the epithelial proliferations in the wall of the gall-bladder.<sup>14, 19</sup> In all cases multiple or serial sections show that the deeper 'glands' are continuous with the surface epithelium and apparently develop from it.

### 2. Neoplastic Change.—

a. The absence of the criteria of malignancy has been considered.

b. Innocent growth (adenoma) may be excluded by the observations : (i) A definite etiological factor—the chronic inflammation—is to be observed in the surrounding connective tissue. (ii) There is no real encapsulation of the epithelial glands ; they definitely grow into the wall of the organ, though without destruction of tissue. (iii) The amount and degree of differentiation of the heterotopic tissue present does not occur usually in growths.



**3. Precancerous Changes.**—It cannot be denied that the epithelial proliferation may, in late stages, give rise to malignant development. There seems no reason to suppose, however, that this would occur more readily or more frequently in cholecystitis glandularis than in ordinary cholecystitis. There has been no suggestion of malignancy in any of the cases which we have observed.

The exclusion of these various hypotheses necessitates their replacement by an alternative. The theory of metaplasia (heteromorphosis) is now on a well-established basis. Tissue of one type often changes to another related form, probably by de-differentiation of the cells at first, followed by re-differentiation. Any related form of tissue may arise. The epithelium of the gall-bladder arises from that of the primitive alimentary canal, and may, given the suitable stimulus, return to a primitive form. When this differentiates once more, it may form, not typical gall-bladder epithelium, but, depending on the stimulus, epithelium resembling the cells and structures resembling those of the stomach, duodenum, or intestine.

Proliferation of connective tissue as the result of chronic irritation is a well-recognized phenomenon, but epithelial proliferation in similar circumstances, though sometimes more important, is often not appreciated.

In several parts of the body, but particularly in the stomach, this epithelial proliferation associated with chronic inflammation has been interpreted as carcinoma or as precancerous, so that very high proportions of gastric ulcers were thought to be malignant. Critical examination has shown this view to be inexact.

The proliferative epithelial changes in the gall-bladder should be regarded as the reaction of the epithelial cells to the chemical stimuli occurring in an inflamed gall-bladder.

### SUMMARY.

1. A number of examples of diffuse or localized thickening of the wall of the gall-bladder which are honeycombed by epithelium-lined crypts and glands are described.

2. Some of these cases conform with the condition described by Bodnár as 'cholecystitis eystica', but since others present the characteristic involvement of the wall without cyst formation, the term 'cholecystitis glandularis proliferans' is applied.

3. Several types, which depend on the site of the epithelial development, are described.

4. They develop as a result of epithelial proliferation with metaplastic change into various types of alimentary canal glands.

5. It is not malignant, and the diagnosis from malignancy is important, but easy when the nature and characteristics of the condition are understood.

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## *VISITS TO SURGICAL CLINICS AT HOME AND ABROAD.*

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### **DR. MURK JANSEN AND HIS CLINIC AT LEIDEN.**

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DR. MURK JANSEN, the leading orthopædic surgeon of Holland, is well known amongst British surgeons as a researcher, philosopher, and practical surgeon (*Fig. 253*). He had the advantage (as every specialist should) of a very thorough grounding in general principles; after over four years' post-graduate work as prosecutor of anatomy, and as an assistant in internal medicine and general surgery, he studied in Vienna and in Würzburg, and practised internal medicine and orthopædies for some years in order to obtain a sound insight into the fundamental principles of pathology and diagnosis. The special study of orthopædies attracted him, as he realized the scope that this specialty offered for original investigation; and, as opportunity offered, he devoted himself entirely to orthopædic work. About this time he was invited to become Lecturer in Orthopædies at the ancient university of Leiden; he took up his abode in that city, and has remained there ever since. He has a few beds in the University Hospital, which is a large state-run institution to which all persons have the right of admission; and he has over sixty private beds in the Anna Kliniek voor Orthopaëdie (*Fig. 254*), which is a private hospital for the treatment of paying patients.

This beautiful little hospital, which will form a model, it is hoped, for all others in Holland, was opened on Nov. 11, 1929. It stands upon the outskirts of the city, in its own grounds, surrounded by an orchard and a garden, and is as perfect as a combination of surgical and architectural skill could make it. Apart from a few patients transferred from the University Hospital, who pay for extras only, all patients pay; they are divided into five classes, paying from 2.50 to 10.50 guilders per day (12 g. = £1), and are housed in private rooms and small wards of two, three, or four beds. A very large, light operating-theatre (*Fig. 255*) and a plaster-room (*Fig. 256*) are provided, and the hospital has its own instrument-shop, which employs thirteen men, and produces all the splints and instruments needed by the patients. The nurses are state-registered, and receive a three-years' training.

Dr. Jansen has a very large hospital and private practice, patients coming to him, not only from all parts of Holland and Belgium, but also from more distant countries. Dr. Fransen, his partner, assists him in all his work, public and private, and sees all out-patients at the University Clinic; he also

performs a number of operations. Dr. Jansen sees private patients daily at his house from 1 to 3 p.m., and his organization for dealing with a large number of patients is very interesting; the histories are prepared by his assistants, the patients are made ready for him to examine in a number of rooms and cubicles, a card-record of each case is placed in readiness on the door, and X-ray and other investigations are made on the spot as required.

Numerous operations are performed; at the Anna Kliniek alone, 1057 operations have been carried out in the main operating-theatre during the past year and a half. The technique and instruments used are practically



FIG. 253.—Dr. Murk Jansen.

identical with those employed in this country. The rubber gloves are dry-sterilized by the vapour of paraform tablets, the various sizes of gloves being stored on metal trays in a cabinet somewhat resembling a microscope-slide cabinet; the tablets are changed every three or four months, and I was told that there has never been the slightest anxiety about the sterility of the gloves prepared in this way. Forty-eight hours' exposure is allowed for the sterilization to take place. Alcohol is largely used for the sterilization of apparatus, and of the operator's skin; and Kirschner's method, with a 7.5 per cent solution of tannic acid in alcohol, is exclusively used for the final cleansing

of the patient's skin, following the usual treatment with soap, spirit, and ether; the tannic acid method is stated to be superior to all others in its efficiency and freedom from production of dermatitis.

Three operations were seen :—

*Operation 1.*—Man, age 67. Bucket-handle tear of semilunar fibrocartilage; no tourniquet used in this particular case because of cardiovascular degeneration. Rather long curvilinear horizontal incision; gauze skin-guards; dry gauze swabs; fibrocartilage excised with a long, narrow, sharp-pointed Adams' knife; catgut ligatures and capsule-suture; linen-thread in skin; wire cradle back-splint for ten days, then active exercises.

Dr. Jansen is a vigorous opponent of the indiscriminate use of massage, considering that far better results are attained at far less cost by the energetic exercising of the patient's muscles by active voluntary exertion. He

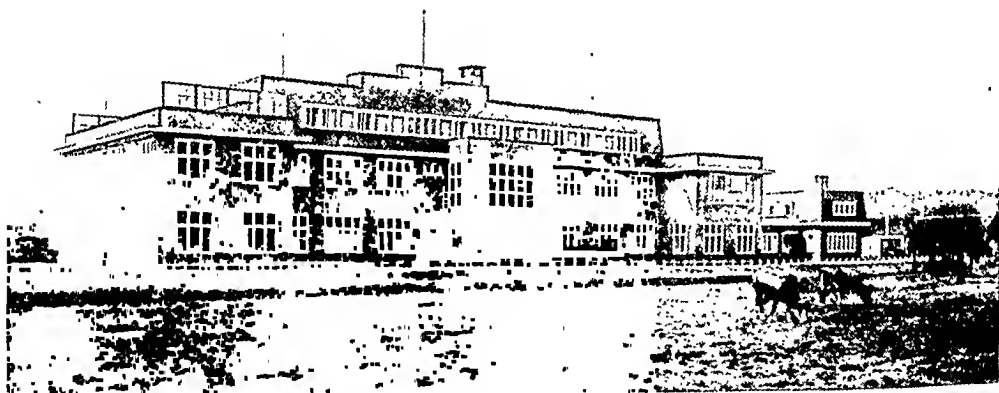


FIG. 254.—Anna Kliniek voor Orthopaëdie, Leiden.

holds that massage and electrical treatment are often exploited unnecessarily, and are foisted somewhat unscrupulously on a gullible public; he uses massage and electricity, of course, where they are definitely indicated, but he lays it down as a general principle that no method of treatment should ever be employed without a clear understanding of its mode of action and of its purpose, and without a clear indication for its use. He believes and teaches that failure to observe this principle is largely responsible for the absence of any striking recent advance in therapeutics.

*Operation 2.*—Woman, age 51. Re-operation for hallux valgus, after unsuccessful operation elsewhere. Infiltration analgesia with about 25 c.c. of 0.5 per cent novocain solution. Base of proximal phalanx removed with very sharp and powerful bone-cutting shears. Triangular flap of capsule, v-shaped, pointing proximally, sutured well back on inner side of toe, to

maintain adduction afterwards; abductor hallucis tendon then sutured to this, after transplantation, to overcome the rotation of the toe which is found always to occur in these cases.

Dr. Jansen said that he found the removal of a good-sized piece of the base of the proximal phalanx (the operation introduced into England by Davies-Colley, and known by his name) was in every way a most satisfactory proceeding.

*Operation 3.*—Woman, age 27. Progressive muscular dystrophy. A standard Naughton Dunn stabilization, combined with a posterior bone-block, after the style of Willis Campbell, was performed. The special feature of this case was the method of dressing immediately after the operation; gauze, about  $\frac{1}{2}$  in. of absorbent white cotton-wool, and an ordinary open-weave



FIG. 255.—The operating-theatre in Dr. Jansen's clinic.

bandage, were applied; then a further layer, at least  $1\frac{1}{4}$  in. thick, of very good non-absorbent wool, and finally a bandage of tubular stockinette applied exceedingly tightly; the tourniquet was then removed. It was stated that this method entirely prevented hæmatoma and post-operative pain, the foot being almost as firm as if it were encased forthwith in plaster-of-Paris. The foot was then placed in an ordinary wire cradle-splint, and plaster would be applied after the skin sutures had been removed in about a fortnight's time; this plaster would be taken off after six weeks, and the patient would then be allowed to walk on a second and final plaster, kept on for two months.

After the operating session, photographs and radiograms of a case of congenital absence of the sacrum ('asaeria') were shown. This rare developmental

abnormality has been observed and described once before by Dr. Jansen. The buttocks are notably small, the anus deep, and the child, if male, sits on the scrotum. The radiogram shows complete absence of the sacrum, and the presence of lumbar spina bifida; the father also has spina bifida. The condition is attributed to smallness of the amniotic sac at an early stage, before the period of infolding.

In the wards were shown a number of interesting orthopædic cases. Amongst these was one of claw-hand, the result of progressive muscular dystrophy, in which arthrodesis of the proximal interphalangeal joint of the fourth finger had been performed in order to restore grip. For claw-toes it is Dr. Jansen's practice, in suitable cases, to arthrodesise the proximal



FIG. 256.—The plaster room at Dr. Jansen's Clinic.

interphalangeal joint in order to allow the long flexors to flex the metatarsophalangeal joint; without arthrodesis, the lumbricals and interossei hyperextend the toes at the basal joints in claw-foot.

Dr. Jansen has wholly abandoned the use of remedial exercises in the treatment of scoliosis and kypho-lordosis, and has substituted for it the wearing of a simple appliance which may be called his 'mental educator'. (The strict translation of its Dutch name is 'straight reminder', the instrument being intended to remind the patient to stand straight.) It consists of a light pelvis-band, to which is riveted an upright metal bar, exerting slight pressure by means of a pad upon the maximum convexity, and having a narrow leather strap around the neck. The patient is thus constantly reminded to make a corrective muscular effort, so as to avoid the pressure of the pad upon the convexity. It is found that after the educator has been

worn for a year or more, marked improvement occurs, and by this means the treatment of scoliosis and kypho-lordosis is enormously simplified and cheapened, especially in the case of patients living at a considerable distance from the clinic. Apart from a plaster-bed for lateral recumbency employed by night for scoliosis, no corset or appliance of any kind is used other than the educator. Dr. Jansen teaches that anything that even marks the skin of the thorax or of the abdomen is bad. He considers that the improvement in posture and in the tone of the abdominal muscles produces a negative pressure in the upper part of the abdomen; and this negative pressure (whose existence has been demonstrated by manometric readings taken with a colonic tube) causes engorgement of the liver and other vital organs with healthy blood. This accounts for the marked improvement in general health that is invariably observed as posture improves.

Another interesting out-patient case was that of a girl aged 10 years, having weak muscles, slight knock-knee, and genu recurvatum, who complained of a sensation as of something becoming caught between her femur and her patella. No foreign body or loose body was present, but a lateral radiogram showed flattening of the femoral condyle, and a sloping of the upper surface of the tibia backward and downward. The epiphysial ends both of the femur and of the tibia appeared to have swung backward, the former upward and the latter downward, so that the joint-space had become narrowed in front. Dr. Jansen explained that this led to the patella becoming unduly high, the fat-pads becoming prominent in front and liable to be nipped; this explained the patient's symptom. He has described this condition as 'patella alta spuria', in contradistinction to 'patella alta vera', in which the patellar ligament is abnormally long.<sup>1</sup>

A number of other interesting cases were seen, some of which gave Dr. Jansen an opportunity of expounding, very lucidly and convincingly, his well-known theory of the causation of a number of obscure developmental abnormalities, such as achondroplasia, osteogenesis imperfecta, etc.<sup>2,3</sup>

On Thursday, May 21, 1931, an interesting little ceremony was performed at the Anna Kliniek voor Orthopaëdie. Dr. Jansen unveiled a bronze plaque of Sir Robert Jones which has been erected on the main staircase of the hospital, and delivered a short address, in which he described the great part played by Sir Robert Jones in the development of the science of orthopaedic surgery. Sir Robert Jones, who was supported by several other British orthopaedic surgeons, made a suitable reply.

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# SHORT NOTES OF RARE OR OBSCURE CASES

## AN EXAMPLE OF BENIGN OSTEOGENIC SARCOMA.

BY E. S. J. KING,

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THE advantages accruing from reporting cases of rare conditions such as bone sarcoma are demonstrated by the conclusions which have been drawn from the specimens in the American Registry. Any example of malignant tumour of bone is worthy of record, and particularly one which illustrates some special feature. For this reason the following case is described:—

M. D., male, age 19, reported for examination on account of a generalized swelling of his left knee of about twelve months' duration. During the four or five months prior to examination the knee had become puffy, and although there had been but little pain, the leg was unable to support the weight of the body. There had been some pain on movement.

ON EXAMINATION.—The left knee was swollen and puffy, with no obvious fluid. The lower end of the femur was slightly tender over the outer condyle, but with little tenderness over the joint. There was no crepitus, and marked limitation of movement, accompanied by pain. All the other joints of the body were normal. The lungs were normal, both on clinical examination and by X-ray. The X-ray of the knee-joint showed erosion of the inner aspect of the outer condyle of the femur. The pre-operative diagnosis was tuberculosis of the joint.

OPERATION.—A distal horseshoe incision was made, and the flap of skin and subcutaneous tissue turned upwards. The patellar tendon was incised turned upwards. The patella was turned upwards. A with the expansion of the vasti, and the patella together were removed (Fig. 257). The upper surface of the tibia was also removed.

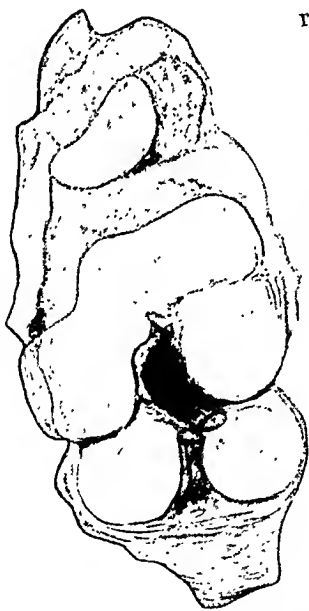


FIG. 257.—Drawing showing the knee open at operation. The small tumour is presenting between the condyles of the femur, arising from the outer condyle.

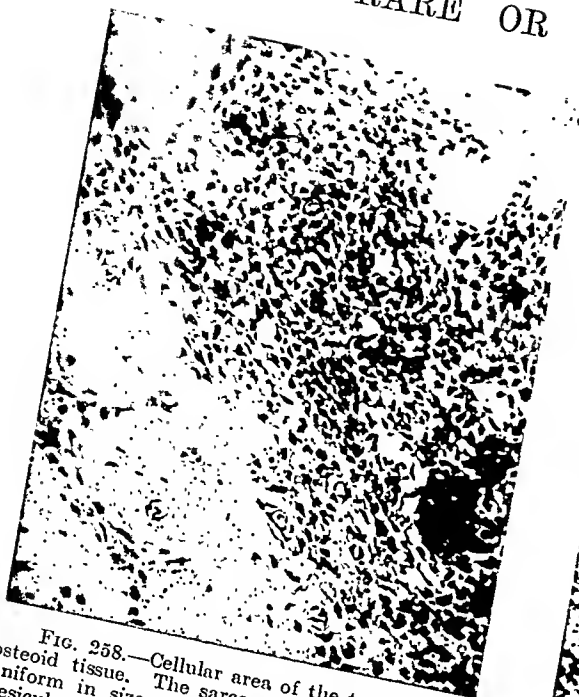


FIG. 258.—Cellular area of the tumour with osteoid tissue. The sarcoma cells are curiously uniform in size and shape and the nuclei less vesicular in appearance than is usual. Three foreign-body giant cells are present. ( $\times 80$ .)



FIG. 259.—Photomicrograph of another area of the growth similar to that shown in Fig. 258, but with a greater proportion of osteoid tissue. ( $\times 80$ .)

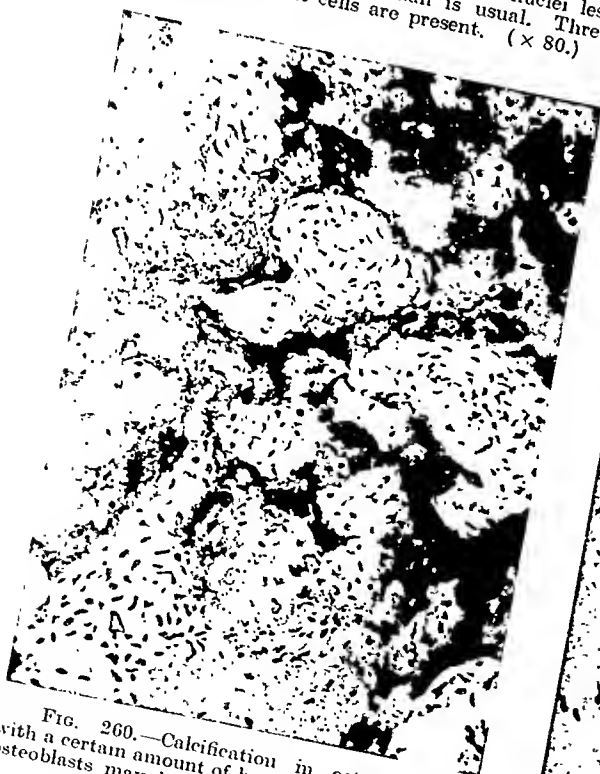


FIG. 260.—Calcification in osteoid tissue with a certain amount of bone formation. A few osteoblasts may be seen. ( $\times 80$ .)

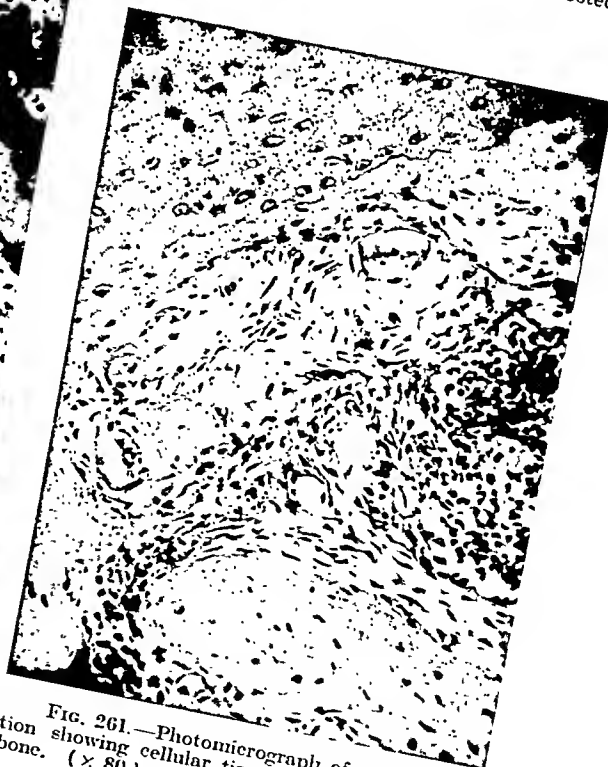


FIG. 261.—Photomicrograph of another portion showing cellular tissue, osteoid tissue, and bone. ( $\times 80$ .)



FIG. 262.—Osteoid tissue, cellular tissue, and cartilage. The cells in this part are more 'active'. ( $\times 80$ .)

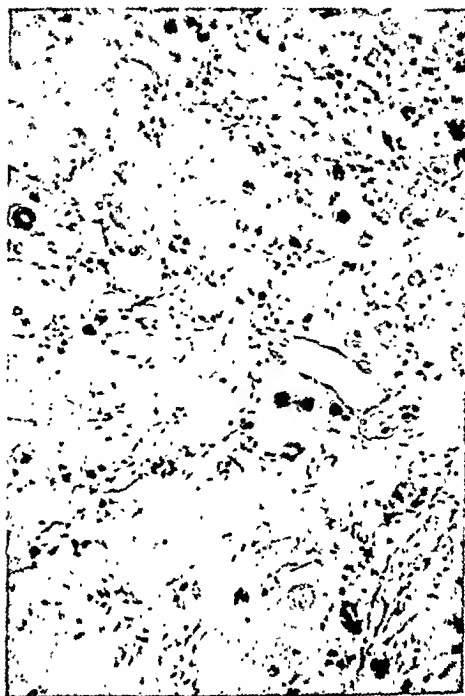


FIG. 263.—Another portion showing actively growing cartilage. ( $\times 80$ .)

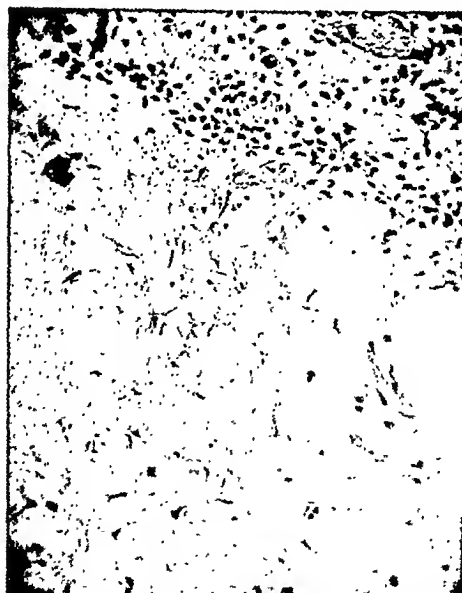


FIG. 264.—Photomicrograph of a section showing cellular tissue merging into mucoid connective tissue. ( $\times 80$ .)

## PATHOLOGICAL EXAMINATION.—

*Macroscopic Appearance.*—The specimen consisted of the outer condyle of the femur and portion of the inner condyle, together with the tumour which was growing from the medial aspect of the outer condyle. The growth measured  $1\frac{1}{2}$  in. in diameter and about one-third of its mass projected from the surface of the bone. It was much softer than ordinary bone, small areas of mucoid change being present, and spicules of bone and calcified material were present throughout the growth.

*Microscopic Examination.*—The growth showed all the characters of an osteogenic sarcoma: (1) Areas of cellular activity, the cells being for the most part of the spindle variety (*Fig. 258*). (2) Formation of osteoid tissue occurred in many areas (*Fig. 259*). (3) Calcification both of the osteoid tissue and of the ordinary tissue occurred (*Fig. 260*). (4) Ossification was present in some areas (*Fig. 261*). (5) Cartilage was present in some portions (*Figs. 262, 263*). (6) The spindle-cell tissue merged into typical mucoid tissue (*Fig. 264*). (7) In some areas many giant-cells were found. (8) In some areas the tissue was more vascular and many cells indistinguishable from cells of the bone-marrow (including myelocytes) were present.

*SUBSEQUENT HISTORY.*—The patient recovered rapidly from the operation, and bone union had occurred at the end of two months. Nine years later the patient is perfectly well—the local condition of the knee region is satisfactory, without sign of recurrence, and the lungs are clear.

## COMMENT.

Though the great majority of primary tumours of bone are malignant, some intensely so, a few examples of benign growths have been recorded.

The case described is of interest since no recurrence followed on even very local removal. The microscopic picture showed that the tumour was a typical example of an osteogenic sarcoma. Such a benign course suggests that the tumour might possibly show some distinct morphological differences from other osteogenic sarcomata. Careful examination of the sections showed that the cells were curiously uniform in size and shape and that giant cells present were of the foreign-body type.

Even had this been noted nine years ago it is doubtful whether the bad prognosis then given would have been modified. It suggests, however, that such benign cases should be carefully examined for some such morphological peculiarity. Information is difficult to obtain concerning these tumours owing to their rarity, and only the careful examination of those which do arise will advance our knowledge.

From the point of view of treatment, the question has often arisen: Is it not desirable to treat most sarcomata conservatively by, at least, amputation close to the lesion? Extensive operations occasionally are followed by a cure—but do not these tumours belong to the variety described here? We know that extensive operations in malignant cases will not cure the condition owing to the presence of metastases in the lungs. On the other hand, the less radical methods of treatment will save unnecessary mutilation in the comparatively benign cases.

## EXCISION OF SUBCLAVIAN ANEURYSM ASSOCIATED WITH CERVICAL RIB.

By WILLIAM BILLINGTON,

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DEFINITE aneurysm of the third stage of the subclavian artery associated with cervical rib is a very rare condition, and excision of such an aneurysm with relief of symptoms must be almost unique.

HISTORY.—The patient was a single woman, 42 years of age, who had enjoyed good health until the onset of her present symptoms. For several years she had complained of pains in the back and under the left shoulder, for which no clear explanation was found. In May, 1930, her doctor discovered a definite pulsating lump above the left clavicle, and she then complained of pain in her left arm, and it was noticed that there was a difference in the timing of the radial pulses. A month later the radial pulse on the left side had disappeared, the arm felt cold, and the pain in it had become severe.



FIG. 265.—Fully developed right cervical rib; left cervical rib (indicated by arrow) incomplete.

ON ADMISSION.—In November, 1930, the patient was admitted into my wards at the Queen's Hospital. She was a poorly-nourished woman, but was organically sound. She stated that in May, 1930, her left arm started to ache and the pain had gradually become worse. It had also extended to the shoulder and the left side of the neck. The pain increased when she was at work and especially so when she lifted heavy weights, such as a pail of water. She had no pain at night, or when the arm was at rest. The fingers of the left hand felt cold and numb, and sometimes they went quite white.

ON EXAMINATION.—A swelling could be felt above the left clavicle. It was elongated, about  $2\frac{1}{2}$  in. in length, and at its centre about the size of a walnut. No pulsation could be felt in it. The swelling could be moved up and down, but only slightly from side to side. When the shoulder was raised the swelling slipped under the clavicle, and part of it could be felt beneath the pectoralis major and internal to the coracoid process. There was no

evidence of median or ulnar paralysis. The radial pulse could not be felt, and the tips of the fingers were cold. No weakness of the arm existed. A Wassermann test was negative.

*X-ray Report.*—Dr. J. Brailsford took an X-ray photograph and reported upon it as follows: "The radiograph shows a fully developed right cervical rib. The left cervical rib is incomplete; its anterior extremity has formed a symphysis with the first rib on a plane with the clavicle, indicated by an arrow (*Fig. 265*). The swelling in the soft tissues is in this area".

*OPERATION.*—A curved incision about 4 in. long was made parallel with and a little above the clavicle in its outer part, and extending vertically upwards at its inner end over the posterior margin of the sternomastoid muscle. The lump was readily exposed and found to be an aneurysm of the subclavian artery. It commenced at the outer edge of the scalenus anticus, where the artery was at a much higher level than is normal, making the long axis of the aneurysm almost vertical. There was no pulsation in the sac. The lowest trunk of the brachial plexus was adherent to the outside of the sac, and moved with it. The vessel at either end of the aneurysm was tied with a double catgut ligature, and the aneurysm was excised. As the cervical rib did not appear to be interfering with the nerves of the brachial plexus, it was not disturbed.

*REPORT ON THE ANEURYSM* (Dr. Neale, Pathologist to the Queen's Hospital).—"The portion of the artery removed is approximately  $2\frac{1}{2}$  in. in length and shows a fusiform dilatation reaching a maximum diameter of  $\frac{3}{4}$  in. At the place of maximum dilatation the external

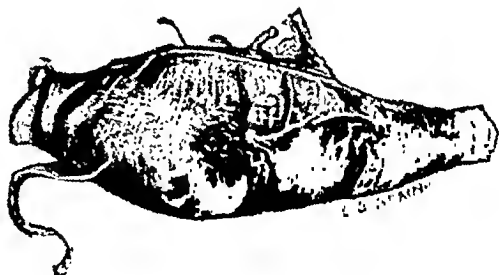


FIG. 266.—The aneurysm.

surface of the artery feels thin, and foci of hæmorrhage are seen in the adventitial tissue. The lumen is largely occupied by a laminated thrombus formation through which, however, a minute channel is detectable. (*Fig. 266.*)

"Microscopically, the muscular coat shows a slight degree of fibrotic change without evidence of inflammation. The internal elastic lamina at one place is duplicated and fragmented, and at this site thrombosis has occurred, showing as a laminated deposition of fibrin—the deeper layers of which show fairly advanced organization and canalization. There is no primary disease in the intimal layer of the artery. The findings are consistent with traumatic injury to the artery."

*SUBSEQUENT PROGRESS.*—The patient made an uneventful recovery, and all pain in the shoulder and arm disappeared. The pain was apparently caused by pressure upon the lowest trunk of the brachial plexus by the sac of the aneurysm, especially when the latter was pulled upon by lowering of the shoulder.

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## A CASE OF SUPPURATIVE PYLEPHLEBITIS SECONDARY TO CARCINOMA AT THE DUODENAL BILIARY PAPILLA.

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AND T. B. DAVIE,

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THE condition of suppurative pylephlebitis with generalized abscess formation throughout the liver, resulting from the obstruction to the opening of the ampulla of Vater by a carcinoma at this orifice, is of sufficient rarity in our opinion to warrant the publication of the following case :—

**HISTORY.**—The patient, a male of 51 years, had been investigated in hospital for two transient attacks of jaundice, each lasting a week, with a six-month period of freedom from symptoms between attacks. During the attacks there was slight aching pain in the right upper quadrant of the abdomen, and investigation showed enlargement of the liver, the anterior edge reaching down nearly to the umbilicus. The liver was smooth, rather soft, and not tender. Examination by Graham's method showed failure of the gall-bladder to fill with dye, and van den Bergh's test was strongly suggestive of obstructive jaundice. However, the relative absence of pain and the success of medical methods in clearing up the jaundice led to the diagnosis of catarrhal jaundice, and he was discharged from hospital. One month later he was re-admitted suffering from severe colicky pain of sudden onset in the upper abdomen, intense jaundice, fever, and with a history of a rigor on the day previous to admission.

**ON ADMISSION.**—Intense jaundice, temperature  $102.6^{\circ}$ , pulse 100 ; liver enlarged to below the umbilicus, slightly tender—more so over gall-bladder area—smooth, and somewhat soft. Gall-bladder not palpable. Epigastric and right hypochondriac tenderness and rigidity. Blood : white-cell count 22,000 per c.mm., mostly polymorphonuclears. Another rigor occurred shortly after admission to hospital. A diagnosis of suppurative cholangitis was made, obstruction to the common bile-duct by calculus being considered probable, and drainage of the gall-bladder was decided upon.

**OPERATION.**—Through a right-sided paramedian incision, centring at the umbilicus, the gall-bladder was exposed. This viscus was found to be markedly distended and thin-walled, and concealed to palpation by the enlargement of the liver. The liver was smooth and intensely and evenly jaundiced, and no abscesses were visible. No calculus was palpable in the gall-bladder, nor in the cystic and common bile-ducts. The pancreas felt firm but not malignant. Continuous with the head of the pancreas was felt a rounded firm mass apparently indenting the second part of the duodenum or protruding into its lumen. Investigations were hurried because of the precarious state of the patient under the anæsthetic. After packing off the peritoneal cavity, a large tube was inserted into the gall-bladder and much foul-smelling pus mixed with 'white bile' was evacuated. The abdomen was

closed, leaving a drainage tube in the right kidney pouch in addition to the cholecystostomy tube.

**POST-OPERATIVE HISTORY.**—Bile commenced to flow from the cholecystostomy tube a few hours after the operation, and continued to do so up to the time of his death five days later. Although the patient felt relieved and his jaundice was considerably diminished, his general condition became steadily worse. Auricular fibrillation supervened two days later and was only with difficulty controlled by digitalis. Uræmia-like symptoms followed, his blood N.P.N. being 120 mgrm. per cent; and death occurred five days after the operation, being preceded by muscular twitchings, anuria, and coma.

**AUTOPSY.**—At the post-mortem examination the enlarged, intensely jaundiced liver was found to weigh 71 oz. Scattered over the surface were numerous small whitish areas, especially noticeable along the anterior edge. These resembled multiple malignant metastases, but proved to be the larger of very numerous abscesses present within the organ, as well as on its surface. Microscopically the abscess formation was associated with the portal tract. In this region the bile-duets, where not involved in the abscesses, were dilated, and some contained pus. The portal veins were never visible, being almost certainly the centres from which the abscesses had spread, i.e., the appearances suggest that the lesion was a pyelephlebitis rather than a pure suppurative cholangitis (*see below*). The duodenum was opened *in situ*, and the mass which at the operation had appeared to be indenting the duodenum at the site of the head of the pancreas was found to be a firm papilliferous growth springing from the site of the biliary papilla (*Fig. 267*). It measured  $2\frac{1}{4}$  in. in the line of the lumen of the gut, and 1 in. across, and projected  $\frac{3}{4}$  in. into the lumen from a broad pedicle. The orifice of the ampulla of Vater was completely obliterated. Histologically the tumour proved to be a columnar-celled carcinoma with a papilliferous surface, arising from the intestinal mucosa lining the biliary papilla.



FIG. 267.—Photograph of the growth *in situ*. ( $\times \frac{2}{3}$ .)

Inflammation within the gall-bladder had subsided considerably following the cholecystostomy, and the whole operation area was free from gross infection. The common bile-duet was only moderately dilated, showed inflammatory congestion only, and did not contain pus; nor was there any obvious suppuration within the portal vein.

The kidneys showed a moderate degree of chronic fibrosis and of jaundice pigmentation. The lungs were affected by a patchy hypostatic pneumonia and marked oedema, the oedema fluid being bile-stained. The pancreas,



though firm and somewhat fibrotic, showed no malignant change or involvement; and the enlarged lymphatic glands found around its head showed acute inflammatory changes only.

### COMMENT.

On the pathological side, the nature of the growth and the intrahepatic suppuration are of interest. The relatively large size of the lesion in this case, and the absence of involvement of the pancreas, rule out true Vaterian carcinoma, as also carcinoma of the duct of Wirsung and of the common bile-duct.

The appearance of the pyelephlebitis within the liver without obvious suppuration in the extrahepatic portion of portal vein, though uncommon, is well recognized. In cases of this nature it is suggested that the primary lesion is an infective cholangitis and that abscesses form outside the ducts, possibly in the lymphatics. From these, suppuration spreads to the branches of the portal vein, whence a pyelephlebitis follows with diffuse suppuration of the portal spaces in the liver.

Clinically the case presents several points of interest. The transient nature of the two earlier attacks of jaundice is contrary to much of the teaching in connection with malignant growths of or near the bile-passages. Rolleston,<sup>1</sup> however, in 1905 drew attention to this point, and stressed the fact that, while growths of the intestinal mucosa covering the biliary papilla were not common, they were particularly prone to produce an ascending infective cholangitis with intrahepatic suppuration. This resulted from, *inter alia*, the obstruction to the bile outflow, which in its turn was liable to intermissions following ulceration and necrosis of the growth. More recently Carnot (1928), quoted by Barron,<sup>2</sup> has similarly stressed the intermittent nature of the jaundice in tumours of the papilla of Vater. The importance of these considerations is obvious, as a recognition of this cause of intermittent jaundice is essential to the diagnostician who would assign a cause to a transient attack of jaundice occurring in elderly patients.

Other factors in this case which helped to cloud the diagnosis were the absence of any history suggestive of cholelithiasis, the failure to palpate the gall-bladder enlargement, and the absence of evidence of involvement of the pancreas. All these observations were in favour of a diagnosis of catarrhal jaundice; and probably the only factor against this diagnosis was the age of the patient.

The clinical course and operative and post-mortem findings in our case agree very closely with the observations of Cohen and Colp<sup>3</sup> (8 cases, 1927) and Wahl<sup>4</sup> (6 cases, 1924). The former of these authors, however, found that when infection of the tumour area occurred acute pancreatitis commonly ensued; and both authors emphasize the frequent occurrence of intestinal hæmorrhages. Neither of these conditions appeared in our case.

In conclusion we submit that a consideration of this case illustrates the following important points:—

1. It is wise to avoid the diagnosis of catarrhal jaundice in middle-aged or elderly patients.

2. Carcinoma of the duodenal mucosa covering the biliary papilla produces jaundice which is frequently characterized by its remissions.

3. While suppurative cholangitis is a frequent complication of these tumours, and cholaemic intestinal haemorrhages are not uncommon, a rarer sequel is an intrahepatic suppurative pylephlebitis.

### SUMMARY.

A case of carcinoma of the duodenal mucosa of the biliary papilla is reported.

The difficulties of the diagnosis of this condition are explained, and the importance of the feature of intermittent jaundice in elderly patients is stressed.

We are indebted to Dr. Bigland and to Mr. Hugh Reid for permission to publish this case.

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## A FIBROMYOMA OF THE STOMACH WEIGHING OVER 2 LB.

BY A. J. BLAXLAND, NORWICH.

APART from its size, this tumour is of interest in that clinically it did not appear to be connected with the stomach, and it caused no obvious symptoms until necrosis developed in a portion of it.

HISTORY.—The patient, a man of 60, sent to me by Dr. Gaynor, of Wymondham, was known to have had a large swelling in the abdomen for at least two years. He had had some dyspepsia on and off for many years. On Dec. 10, 1930, he developed abdominal pain and pyrexia.

ON EXAMINATION.—Abdominal examination revealed a solid, semi-elastic, tender, rounded swelling, about the size of the head of a year-old child. Its centre was 2 in. to the right of the umbilicus and 1 in. below it. It was almost immobile.

OPERATION (Dec. 19).—Coeliotomy was performed. The tumour was found to lie in the lesser sac. The transverse mesocolon was adherent over an area on its lower aspect, where the wall of the tumour had become gangrenous (and a little stinking pus escaped on undoing these adhesions). Apart from this the tumour lay free in the lesser sac, except where it was attached over a width of 2 in. to the posterior wall of the stomach. An elliptical incision through the stomach wall around this attachment completed the removal of the tumour. Except for some suppuration in the lower end of the wound the patient made an uneventful recovery.

**PATHOLOGY.**—The tumour is now in the Museum of the Royal College of Surgeons. Sir Arthur Keith informs me that it is the largest specimen of its kind in the Museum. It weighs 2 lb. 6 oz., and is 7 in. across in its longest diameter.

Microscopic section confirms the naked-eye diagnosis of fibromyoma. The tumour was solid except for a small cavity in its centre, which communicated



FIG. 268.—Drawing of vertical section through the tumour, showing cavity in centre of growth. The arrow points to the attachment of the tumour to the posterior wall of the stomach, and shows the mucous membrane dipping down into the growth. The dark portion of the left-hand side of the growth is an area of necrosis. ( $\times \frac{2}{3}$ .)

with the lumen of the stomach by an orifice  $\frac{1}{2}$  in. in diameter; and the mucous membrane of the stomach can be seen dipping down into this orifice (*Fig. 268*). In relation to the stomach wall the fibromyoma was entirely subperitoneal. The dipping in of the mucous membrane suggests that at an early stage of its growth the tumour was partly submucous and protruded into the lumen of the stomach.

## REVIEWS AND NOTICES OF BOOKS.

*Nasenplastik und sonstige Gesichtsplastik, nebst einem Anhang über Mammaplastik und einige weitere Operationen aus dem Gebiete der äusseren Körperplastik.* By Prof. Dr. J. JOSEPH (Berlin). Imperial 8vo. Pp. 843 + xxxi, with 1718 illustrations, many in colour. 1931. Leipzig: Johann Barth and Curt Kabitzsch. RM. 242.

THIS monumental work is notable for its size, completeness, and the number and beauty of its illustrations. It begins with eighty photographic illustrations of the typical forms of defect in the nose, face, and breast, the treatment of which forms the bulk of the volume. A short chapter on the anatomy of the face is followed by a critical analysis of the face profile, giving angles and measurements which represent normal and abnormal variations. The profile of the nose is made up of three parts: the upper by the nasal bones, the middle by the septum and main nasal cartilages, and the lower by the nasal tip. Any one of these three parts may be altered by injury or disease in the direction of failure or excess, and forty-one outline drawings are given of the possible variations in the profile of the nose. A short section on biological principles of the survival and growth of transplants, both skin and bone, is followed by the most important part of the book dealing with the fundamental principles of plastic surgery.

In the majority of the operations anæsthesia is obtained by the use of local infiltration by a 1 to 2 per cent solution of novocain combined with adrenalin, but general inhalation anæsthesia is used for children and nervous adults. As regards apparatus and instruments, the technique is remarkably simple and unencumbered with special tools. There are, of course, specially shaped saws and raspatories for intranasal use, but for the taking of bone-grafts for the face a simple bow saw or a Gigli saw is used.

One of the most interesting chapters is that which describes the re-shaping of large or ugly noses, and this part lends itself especially well to the most intriguing illustrations of how ugly people are made beautiful. The large humped nose is reduced by subcutaneous and submucous resection of either the bridge or the nasal walls. Deformities of the alæ and the tip of the nose are corrected by excision of or addition to the cartilage of these structures. Loss of the whole or part of the nose is by far the most important problem of nasal plastic surgery. A full historical account is given of this subject with a short review of the various methods which have been used up to the present day. We think that in this and in subsequent sections the methods of tube-flap which the work of Gillies has made so familiar to us are not recognized as fully as they deserve. For the most part the methods advocated are those of the pedicle flap from the arm, which is recommended chiefly for women and in cases in civil practice; and the pedicle flap from the forehead, which is more suitable for men and for war surgery.

It is quite impossible to do justice to a work of this size in a short review, and one can therefore only touch upon one or two outstanding points. In the reconstruction of total loss of the nose, there are three main structures to be provided: the septum is provided by a free graft from the tibial crest, which is first implanted into the lip and then raised into position; the lining of the nose is provided by cheek-flaps; and the outer skin by the classical flap from the forehead. In cutting the tibial graft a bow saw is used, and it is remarkable how very little in the way of special instruments is recommended.

The remainder of the work deals with the plastic operations for damaged eyelids, lips, and cheeks, the operation for facial paralysis, the reconstruction of the jaws, the external ears, and the raising and reducing of hypertrophied pendulous

breasts. In facial paralysis, the usual nerve anastomosis operations are described, but also some ingenious plastic devices for supplementing the facial muscles by slips taken from the temporal and masseter muscles. In the reconstruction of the proximal part of the lower jaw, one of the metatarsal bones is used, making use of the head of the latter as the condyle of the jaw. To make good the loss of the external ear, a model is made of thin ivory, perforated with many holes. This is implanted in suitably placed skin-flaps which are finally swung into position.

The completeness of this work on plastic surgery, and the great wealth of illustrations make it an invaluable book of reference, and afford a wonderful testimony to the industry of its author and the high standard of its publisher.

Collected Papers 1904-1929. By EDWIN BEER, M.D. (New York). Large 8vo. Pp. 827 + xii, with 252 illustrations. 1931. New York: Paul B. Hoeber Inc. \$7.50.

PERHAPS the most remarkable feature of this book is the diversity of subjects which the author has studied during the period under review, such as the causes of gall-stones, the pegging of fractures, the removal of a foreign body from the bronchus, and a long study of urinary diseases. The majority of the subjects are treated in a masterly manner. It is notable that there is perhaps an element of closer personal contact with the literature in the earlier papers than in some of the later ones. In general it may be said that there is evidence of a greater expenditure of time on the part of the author in the earlier papers than he has found possible to give to the later ones. For instance, the chapter on diverticula of the intestine (1904) is informative, well constructed, and deals with all aspects of the subject. Many of the author's own experiments are recounted. By comparison the paper on chronic tubercular kidney (1921) is rather more in the nature of an article in a text-book. One advantage of the book is that the bibliography fills the gap so often missing in more recent articles on surgical subjects. The chapter on the significance of diarrhoea following abdominal operations (1923) calls attention to a subject of clinical interest and value. Although well known and taught to students many years before the author's first observations of it, it is none the less profitable to read. The consideration of intrahepatic cholelithiasis in another chapter is well conceived.

One great value in any collection of papers is that the progress of any particular subject in the author's mind will frequently reveal its position in the profession as a whole, especially when the papers have been read in the opening of discussions at societies. Such papers as those on cryoseopy and on renal tests in general should perhaps have been referred to and correlated with the author's more mature experience in the 1928 paper. Had he given us from the many thousands of tests which he has performed his definite conclusions as to whether the nitrogenous excretion goes hand in hand with the excretion of dyes he would have greatly added to our knowledge and enabled us to retain the essential tests and discard the others. He writes, "The student of functional tests, however, appreciates the dangers which he is facing, whereas prior to these tests there was failure to realize how thin the ice was." The animal experiments on renal function (1912) are particularly interesting, and technically the author's method of catheterizing both ureters in a child at the same time by employing a single catheterizing cystoscope and two whalebone guides is ingenious. One would like to have his experience of the method since the date of publication (1911), for, as so often happens, it may be impossible to pass even a small cystoscope when there is a catheter already present in the urethra.

There is, as may often happen in a collection of papers, a large amount of repetition—for instance, 167 pages are devoted to tumours of the urinary bladder. In these an illustration of the use of a rubber hot-water bottle as part of the drainage system is repeated on pages 364, 382, and 462, and diagrams of currents used for coagulation and illustrations of the technique of removing growths of the bladder are often repeated. Were it possible, in a future edition, to replace these chapters by one arranged in chronological order quoting the articles and setting forth the author's views at different periods of his experience, repetition would be replaced

by brevity and crudition. The author's enthusiasm for surgery is evidently not waning, judging from the manner in which in 1926-8 he sets forth the development and progress of the surgery of the spleen and associated blood diseases.

Throughout the work the illustrations are clear and well reproduced. The patient applying a hot-air douche for sciatica appears to be turning it away—perhaps because it was too hot! The typography is clear, the style often forcible and always lucid, misprints are few, but the index is insufficient. There is a whole chapter dealing with lime deposits in the kidneys, yet the words 'lime', 'chalk', or 'stone' are not mentioned in the index. This collection of papers as a whole is excellent and well repays perusal.

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**Minor Surgery and Bandaging for the Use of House Surgeons, Dressers and Junior Practitioners.** By GWYNNE WILLIAMS, M.S., F.R.C.S., Surgeon, University College Hospital. Twentieth edition. Crown edition. Pp. 445 + viii, with 262 illustrations. 1930. London: J. & A. Churchill. 10s. 6d. net.

WHEN a book has reached a twentieth edition it is quite superfluous to extol its merits, which in this case have been appreciated more and more since its first appearance in 1861. On the other hand, one feels more justified in making criticisms with the hope that the character and usefulness of the work may be still further improved. Our first criticism is to express surprise at the original authority of the book having been suppressed. Surely a book which was planned and written by Christopher Heath should still show his name somewhere on the title-page?

The present edition has been revised throughout, particularly in the chapters about the treatment of fractures and the administration of anaesthetics. The descriptive matter on the treatment of fractures is clear and sound, but for an up-to-date book we think more care should have been taken with illustrations. For example, *Fig. 181* is not Hey Groves's, but Borchgrevink's, humerus splint, and is probably quite obsolete. *Fig. 209*, showing the suspension treatment of fractured femur in a child, depicts each leg fixed to a back splint and the two back splints fixed to each other. Surely the effect of suspension will be much more efficient if no splints are used? Skeletal traction can be applied by many methods, but why choose the least simple and efficient of these (*Figs. 215 and 216*) in depicting long screws driven into the bones, rather than the transfixion pin or the taut wire? Of all the Thomas splints, those for the hip and the knee figured in *Figs. 237 and 241* are almost obsolete, but the walking caliper splint is not mentioned. In the section on anaesthetics, we think that undue space is given to chloroform and to the Vernon Harcourt apparatus. All these points are, however, only minor matters, and we have no doubt that the book will continue to be the *vade mecum* of the present and future generations of dressers and house surgeons.

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**Surgery: its Principles and Practice. For Students and Practitioners.** By ASTLEY P. C. ASHURST, A.B., M.D., F.A.C.S., Professor of Clinical Surgery in the University of Pennsylvania. Fourth edition, thoroughly revised. Large 8vo. Pp. 1189 + xii, with 1063 illustrations and 15 coloured plates. 1931. London: Henry Kimpton. 45s. net.

THIS new edition of the well-known American text-book follows closely on the third edition which was reviewed scarcely three years ago in these pages. It may be compared with the new *Rose and Carless*, which it resembles closely except that it is somewhat less bulky—1100 pages as against 1500. The text is clear, concise, and dogmatic. The illustrations are photographic and for the most part original. Among re-written matter there are interesting sections on myelomata, injection of varicose veins, thrombo-angiitis obliterans, spinal anaesthesia, diathermy, hyperthyroidism, etc. The information is therefore quite recent and sound. The chapters on orthopaedic surgery are especially full. It is deservedly one of the most popular text-books for American students.

**Thomson and Miles' Manual of Surgery.** By ALEXANDER MILES, M.D., LL.D., F.R.C.S. (Ed.), Consulting Surgeon, Royal Infirmary, Edinburgh; and D. P. D. WILKIE, M.D., F.R.C.S. (Ed. and Eng.), Professor of Surgery, University of Edinburgh. Vol. I. General Surgery. Eighth edition. Crown 8vo. Pp. 574 + xvi, with 176 illustrations. 1931. London: Oxford University Press. 12s. 6d. net.

IN welcoming the eighth edition of this well-known text-book of surgery, the most important change to be noted is that Professor Miles has Professor Wilkie associated with him as joint editor of the work. The task of editing such a text-book and seeing that its various sections are kept up to date can be no light one, especially when the book has for many years been associated with the Edinburgh School and its traditions, and the sacrifice of a section of doubtful value might be considered sacrilegious disregard for hallowed memories. It is stated in the preface that the editors have endeavoured to avoid adding to the size of the work, and doubtless were it not for that endeavour the book would be very much larger even than it is.

Yet it would appear that if it were possible to remodel the whole work it could be contained in a smaller compass. A great many chapters at present contain material that has been dealt with in other sections, and this applies especially to pathology. Every contributor in dealing with his own subject must give an account of the pathology of the conditions the clinical course of which he is about to describe. This being so, the introductory chapters need to contain only the fundamental outlines of pathology, and no therapeutics, leaving the details of pathological processes occurring in the special tissues and their treatment to the sections dealing with the particular organs themselves. This would prevent overlapping and avoid confusion, and there would be no fear that the student would grow up ignorant of pathology, for he has now become accustomed to seek for special information in this vast field in text-books devoted to the subject.

This criticism may be more readily understood if examples of such redundancies are mentioned. In the introductory chapters dealing with acute inflammation, acute infections of the skin and their treatment are described in detail, and they are again set forth in the chapter on diseases of the skin. Incidentally the processes of the formation of a boil and the formation of a carbuncle are not distinguished, a carbuncle being described as a collection of boils. In the chapter on tumours, the tumours of bone are described, and the descriptions are repeated under diseases of bone, even the atypical skiagram corresponding to the atypical pathological specimen being used to illustrate the account of benign giant-cell tumour!

To show how a student may be confused by two accounts of the same thing, we may quote from the chapter on gangrene: "The occlusion of the vein as well as the artery (by ligature) is not found to increase the risk of gangrene"; and from the chapter on injuries of blood-vessels, "When ligation of the artery is necessary, the risks of gangrene are diminished by ligation of the corresponding vein". Repetition is unavoidable in a work composed of the writings of several contributors, and we sympathize with the editors in their thankless task of choosing that which is more excellent.

The standard of the work as a whole is as high as ever, the descriptions of tuberculous disease and of burns, and the general account of the inflammatory diseases of bone, being perhaps especially noteworthy. The book retains its well-known form, and will easily retain also the affection of those who have grown up and are growing up with it.

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**Crippled Children: their Treatment and Orthopedic Nursing.** By EARL D. McBRIDE, B.S., M.D., F.A.C.S., Instructor in Orthopedic Surgery, University of Oklahoma Medical School. Large 8vo. Pp. 280, with 159 illustrations. 1931. London: Henry Kimpton. 15s. net.

THE care of crippled children requires the services of a composite team including surgeons, nurses, physiotherapists, and social workers. Dr. McBride has written a simple, non-technical manual designed primarily for nurses and other lay assistants. It should be a very useful book, for it keeps to simple and sound principles, avoiding details of particular methods in which the work of one surgeon may well

differ from that of others. English surgeons need feel no hesitation in recommending this book to their nursing staffs and other lay assistants; for, although it is written by an American, the methods of treatment considered differ little from those in common use in English orthopædic hospitals and clinics. The illustrations give examples of most of the common crippling conditions of childhood. They are good in themselves and well reproduced—in fact, the book is open to practically no criticism and serves the purpose for which it is designed in an admirable manner.

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*Die Chirurgie. A System of Surgery.* Edited by Profs. M. KIRSCHNER (Tübingen) and O. NORDMANN (Berlin). Fasc. 30 (Index). Royal 8vo. Pp. 219 + vii. 1930. Berlin and Vienna: Urban & Schwarzenberg. Paper covers, RM. 12; bound, RM. 14.

THIS is the last section of a great work and is entirely a series of indexes. The first is a list of the sixty-six contributors to the work. The next is a register of the contents of the six volumes. This register occupies 58 pages and deals with the contents of nearly 9000 pages. Lastly there follows an alphabetical index of subjects and authors, occupying 149 pages.

Each section of this work has been reviewed in these columns, and it only remains here to offer our warm congratulations to the editors, contributors, and publishers for the production of what is probably the largest and most authoritative work on surgery ever produced. It is, of course, a German work, and for that reason it has merits and drawbacks of its own. It forms a most valuable reference work, representing German practice and teaching, but we think that it does not give an equally authoritative exposition of French, American, and British surgery. But to put against this limitation, there is the fact of the very extensive bibliography which accompanies every chapter. The exposition of the theory of surgery and its pathological basis is perhaps the outstanding feature of the book, rather than the practical discussions on treatment. Our greatest admiration is for the team spirit, which has enabled so many to collaborate in producing such a monument of industry and research.

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*Operative Chirurgie der Knochenbrüche.* By Professor FRITZ KÖNIG (Würzburg). Vol. 1. Operationen am frischen und verschleppten Knochenbrüche. Royal 8vo. Pp. 194 + vi. Illustrated. 1931. Berlin: Julius Springer. Paper covers, RM. 27; bound, RM. 29.80.

THIS is a short and concise work, well illustrated, and written by a master hand of great experience. In regard to the vexed question of the indications for open operation, the author protests against the commonly accepted teaching that the procedure should be reserved for cases in which more conservative methods have failed. But he refers in his argument to those cases of mal-union in which inefficient methods have been persisted in far too long. Well-applied skeletal traction ought to give full length, good alignment, and sufficient apposition within ten days of its initiation, and then if it has failed there is no hindrance to open operation, but rather a great help. We feel sure that this is the solution of this difficult problem. For anaesthesia, local infiltration with  $\frac{1}{2}$  per cent novocain solution is recommended for the majority of recent fractures, but general anaesthesia is used for the fractures of the femur.

The bulk of the book is occupied by detailed descriptions of operations upon special fractures, and follows very closely the methods of plating with which Lane's work has made us familiar. The author discusses on very sound lines the question of the influence of foreign bodies buried in the tissues. It is not so much the danger of sepsis that is to be feared as the interference with the circulation in the bone and periosteum. Nevertheless the great bulk of the work is occupied with a description of plating operations, whereas removable agents—e.g., transfixion pins—or absorbable agents—e.g., bone-pegs—are hardly mentioned. We shall await with much interest the further volume dealing with old mal-united or ununited fractures.



*Verlauf der wichtigsten Knochen- und Gelenkerkrankungen im Röntgenbilde. Eine anschauliche Prognostik.* By Privatdozent Dr. med. VICTOR HOFFMANN, Oberarzt der chirurgischen Universitätsklinik im Augusta-Hospital zu Köln. English and German text. Imperial 8vo. Pp. 264 + x, with 584 illustrations. 1931. Berlin: Julius Springer. Paper covers, RM. 66; bound, RM. 69.80.

THIS work is one of outstanding merit, and the fact of its presentation in English as well as German makes it of great value to us for purposes of reference. A series of 156 severe cases of bone and joint disease is presented, and of each case three to five X-ray pictures are given at different stages showing the progress of the disease. The subjects dealt with are: acute and chronic osteomyelitis, both hæmatogenous and traumatic, new growths, nutritional and metabolic diseases, rheumatism and osteo-arthritis, fractures and their complications, bone-grafting, and arthroplasty.

We have nothing but praise for the book, which in a small compass puts together in a graphic form the whole story of bone and joint disease. We hope that in further editions a greater space will be devoted to the subjects of tuberculous disease of the spine and larger joints, which present themselves in such great variety as to require more examples.

It would perhaps seem ungracious to criticize the English text, which is perfectly clear and intelligible, but it would be worth while to have this revised by an English surgeon so as to make it conform with English phrasing and wording. For example, we speak of the 'involucrum' and not of the 'coffin', of 'arthroplasty' and not 'joint plasty', and so on. We think the book is one which every surgeon should have for reference.

*Injuries to Joints.* By Sir ROBERT JONES, Bart., K.B.E., C.B., Ch.M. (L'pool), F.R.C.S. (Eng., Irel., and Edin.), F.A.C.S., Emeritus President, British Orthopædic Association, etc. Third edition. Pott 8vo. Pp. 195. Illustrated. 1930. London: Humphrey Milford. 6s. net.

WE are glad that this small book of wisdom remains with us and is brought up to date. It was born in the war (1915), and the first two editions have had to be reprinted ten times. The value of the book consists in the two facts of the experience of its author and the essential simplicity of its teaching. The present edition has been revised and brought up to date; the section on ischæmic paralysis has been rewritten. In this revision the author acknowledges the help of Mr. Harry Platt.

*Chirurgia del Dolore. La Chirurgia della Innervazione periferica del Simpatico.* By Prof. IGNAZIO SCALONE. Medium 8vo. Pp. 254 + xvi, with 99 illustrations. 1931. Milan: Ulrico Hoepli. L. 30.

THE surgery of pain is a difficult subject upon which to write a good book. There is wheat in Professor Ignazio Scalone's pages, but it is difficult to sift it from the chaff. The main thesis seems to be that peri-arterial sympathectomy has failed of its promise because perineural sympathectomy, its true counterpart, has been neglected. The author advocates treatment of nerve-trunks just as Leriche treats arteries. He admits that the anatomical, histological, and physiological bases of his technique are still rather nebulous. A very considerable part of the work recapitulates the research and practice of other surgeons, and records experiences differing but little from the generality.

With Raynaud's disease, and the various forms of arteriosclerosis and end-arteritis, Professor Scalone has had a certain success, some equivocal amelioration, and some failure, very much where others have found them. He has an interesting chapter devoted to causalgia and like states; his descriptions are reminiscent of the pages of Madame Athanassio-Benisty (some of whose figures he reproduces with acknowledgement, though her name does not appear in the bibliography) written during the prevalence of war injuries of nerves.

In his eighth chapter the author deals with various forms of pelvic neuralgia of uncertain type; his anatomical elucidations seem rather fanciful, and his case

records carry little conviction. The section is open to the criticism, equally applicable to the subject of the next chapter, renal neuralgia, that the 'neuralgie' element is inseparable in most instances from organic disease of the parts, which calls for and receives treatment at the same time; whilst diagnosis, at the early stage he considers desirable for cure to be likely, must be very difficult and uncertain. In some sections—for example, that on acasia—the description of the sympathetic innervation concerned is followed by nothing specifically related thereto in the operations described—nothing more, that is, than the interference incidental to the operations ordinarily performed. A chapter is devoted to speculations on the part played by inflammation of the sympathetic system in obscure painful abdominal states. To technique is devoted half a dozen pages. Forty-four cases are described at length, but some of them have no very close relation to the subject-matter.

The sub-title of the book is "The Surgery of Peripheral Sympathetic Innervation". Afferent sympathetic fibres are traced from the periphery to blood-vessels and thence to the trunk of mixed nerves; the efferent nerve paths are figured as, in the main, avoiding these trunks. It is understandable, therefore, that the author should emphasize the importance of dealing with such efferent fibres in the adventitia of the main nerve-trunks, and have less to say about attack on the ganglia rather than the vascular adventitia, which has been the recent tendency of many surgeons.

Considering the difficulty any one surgeon has in getting enough of these rare cases of his own to form an adequate body of personal documents, it would be unfair to close without praise for the patience and enthusiasm that have gone to the production of this book, or to leave the impression that it will not be of considerable value to those with enough clinical experience to discern the sound work from the padding.

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**Practical Methods in the Diagnosis and Treatment of Venereal Diseases for Medical Practitioners and Students.** By DAVID LEES, D.S.O., M.A., M.B., D.P.H., F.R.C.S., M.R.C.P. (E.), Surgeon in Charge of Venereal Diseases, Royal Infirmary, Edinburgh, etc. With an Introduction by WM. ROBERTSON, M.D., F.R.C.P., D.P.H., Late Medical Officer of Health, Edinburgh. Second edition. Crown 8vo. Pp. 634 + xx, with 87 illustrations. 1931. Edinburgh: E. & S. Livingstone. 15s. net.

THE fact that a second edition of this work has been called for so speedily indicates that it appeals to a definite and appreciative class of reader—the class that requires a full and accurate account of these diseases, clear indications as to treatment, and, above all, reasoned grounds on how one may arrive at a reliable prognosis. Dr. Lees's book supplies all these desiderata. It is a typical example of what one would expect from the Edinburgh School of Medicine.

The author does not think he can cure primary Wassermann-negative syphilis in six months. He insists on the traditional two years of the combined arsenic-bismuth (or mercury)-iodide treatment. Nor does he utterly condemn mercury as useless, like many of those trained in the Continental school of thought. This is typical of his cautious attitude towards recent and untried opinion.

Naturally no two persons' experience or opinions ever quite coincide. When speaking of expectant mothers, we find him writing: "It is advisable to give such infected mothers the benefit of the treatment in every subsequent pregnancy." We would like to substitute for 'advisable', 'imperative'. We note with approval his remark that it is the cases of neurosyphilis which practically never have any discomfort after lumbar puncture; but we are somewhat surprised at the warm manner in which he advocates tryparsamide in this condition, as our experience has not been so happy. We are also surprised to find that in malariatherapy, he allows only ten to twelve rigors to occur before checking with plasmoquin. Few alienists are now satisfied with less than three or four courses of rigors, checked by intervals produced by quinine.

Turning to the treatment of gonorrhœa, we note with pleasure that the author advocates perineal incision in prostatic abscess, instead of the old barbarous practice of rupturing it into the prostatic urethra with a sound (a line of treatment which can be guaranteed to keep up the discharge for many months); but we regret to find

that he thinks a peri-urethral abscess should be opened into the urethra, and not by incision through the skin. On page 540 the text suggests, probably inadvertently, that stricture of the membranous urethra is common as a complication of gonorrhœa, whereas it is practically non-existent except as the result of trauma. The remarks on the fetish of douching in gonorrhœa in women, and on the over-emphasis of diathermy as a curative agent, are very salutary.

In a work in which so many figures occur there are bound to be errors, even with the most careful proof-reading. Most of these will be obvious to the reader, but the statistics on page 271 seem quite incomprehensible.

Finally, a word about the illustrations. These are frankly disappointing. The essential parts shown are too small or too blurred to be of much use. Instead of 'close-ups' of the lesions, we have half the bodies of the patients. Here we touch on what appears to be an essential difference between the British and the American publisher. The American publisher sees that he gets good scale illustrations, either from a skilled professional artist or photographer. He produces the work superbly; and after one has bought it, tempted by the illustrations, one often finds the text quite unworthy of its setting. We appear to err in exactly the opposite way. The text is generally beyond reproach in English medical works, the illustrations are often quite inadequate.

Dr. Lees has produced a work which is accurate, balanced, and well written. It is a mine of up-to-date information on all things venereal. We regret that he has not been better served by his illustrators, and hope that when the next inevitable edition comes along, he will see that more justice is done to his admirable letterpress.

*Die Avertinnarkose in der Chirurgie.* By Professor D. W. ANSCHÜTZ, Direktor der Chirurg. Klinik, Kiel; Dr. K. SRECUT, Assistent der Chirurg. Klinik, Kiel; and Priv.-Doz. Dr. Fr. TIEMANN, Assistent der Mediz. Klinik, Kiel. Royal 8vo. Pp. 200, with 9 illustrations. 1930. Berlin and Vienna: Julius Springer. RM. 16.50.

THIS book is written by two surgeons and a physician. It is the first attempt to crystallize the experience of surgeons in Germany in book form, and is written with characteristic national thoroughness. The earlier chapters deal with the history and development of avertin narcosis, and with the chemistry and pharmacology of the drug, the latter section being exhaustive. In discussing dosage, the authors are careful to point out that there is no invariable rule-of-thumb method for calculating the correct dose from the body weight. They consider the real criterion to be the metabolic rate of the patient, taking into consideration such factors as age, amount of fat present, nervous state, and so on. The intravenous method of administration is discussed, but is not recommended, the method for most cases being the giving of a single dose per rectum.

A chapter is devoted to the discussion of the degree of narcosis to be aimed at, and the conclusion of the authors is that in 50 to 60 per cent of cases at the present time full anaesthesia can be obtained. The ultimate aim is to produce this in all cases, and the authors express the hope that with refinements of technique this will be possible before long. This is a conclusion with which most anaesthetists in this country will not agree, holding that basal narcosis is the only result which is desirable and safe. It must be remembered that in most German centres anaesthetics as a speciality does not exist, and that these are therefore the ideas of surgeons who are either responsible for their own anaesthetics, or who are deputing the task to more or less unskilled assistants. In those cases in which supplementary anaesthesia is required, the authors regard ether as the anaesthetic of choice, and have no personal experience to give of gas and oxygen.

Finally, 103 cases are analysed in which death followed the giving of avertin. In 74 of these cases avertin is entirely cleared of blame for the fatal result, while of the remaining 29, 17 are described as very doubtful and 12 as possible. The authors profess themselves unable to draw any conclusions from these figures, but assess the mortality-rate at 1 in 10,000 cases. In all these cases the dosage used was considerably in excess of that generally employed in this country. A short section is

devoted to the use of avertin in tetanus, with apparently encouraging results. There is no mention of the use of avertin in obstetrics, a subject on which several papers have appeared in Germany.

*Le Traitement médico-chirurgical de l'Occlusion intestinale aigue et subaiguë.* By PIERRE MOIROUD (Marseilles). Crown 8vo. Pp. 122. 1931. Paris: Masson et Cie. Fr. 18.

THIS small brochure of 120 pages is one in a series "Médecine et Chirurgie Pratiques", published by Masson et Cie. It is in the form of a rather expanded paper without illustrations, and it reviews the more recent work on the indications, technique, and results of acute intestinal obstruction; hernia and cancer of the bowel are excluded. A very complete account is given of obstruction by bands, intussusception, gall-stone, volvulus, Meckel's diverticulum, etc., but the main interest of the book lies in the discussion on paralytic ileus. This most distressing sequel to an abdominal operation is fully considered by the author in the light of modern research. He has arrived at definite conclusions which should be borne in mind by all surgeons. These conclusions are as follows: (1) The value of an abdominal X-ray without barium; (2) The importance of re-hydration of the patient; (3) The re-chlorination of the blood by intravenous hypertonic saline (20 c.c. of a 10 per cent saline every four hours); (4) Lavage of the stomach; (5) Serotherapy by anti-Welchii serum; (6) The mechanical emptying of the greatly distended small bowel by aspiration; (7) The great importance of spinal anæsthesia. The book is most clearly written, and is well worth perusal by all abdominal surgeons.

*Der appendicitische Anfall: seine Ätiologie und Pathogenese.* By LUDWIG ASCHOFF (Freiburg-i.-Br.); mit einem kurzen Beitrag über die Lymphgefäßverhältnisse am menschlichen Wurmfortsatz, by Dr. H. SENG. Medium 8vo. Pp. 125. with 36 illustrations. 1930. Berlin and Vienna: Julius Springer. Paper covers, RM. 12.40; bound, RM. 14.

THIS short discussion on the etiology and pathology of appendicitis by the distinguished German pathologist is perhaps disappointing because of its limitations. We had hoped to find some new suggestion concerning the still unexplained incidence of the disease. The monograph, however, is full of valuable information and very good illustrations. Dr. Seng gives some new and remarkable pictures of the lymph-vessels of the appendix.

A large proportion of the book is occupied in a discussion of the bacteriology of appendicitis which does not seem to have any very practical bearing. An interesting estimate is given of the time periods occupied by the different phases in appendicitis. The primary infection takes twelve hours, ulceration occurs in twelve to twenty-four hours, phlegmonous inflammation following ulceration matures in twenty-four to forty-eight hours, and perforation or gangrene are events which occur after the forty-eight-hour period. Great stress is laid on the frequency of latent infection of the appendix, and for this reason it is advised that this structure should always be removed if possible when the abdomen is opened for any other purpose.

## BOOK NOTICES.

*[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.]*

*Schmerzverhütung: Zwölf Vorlesungen.* By Dr. FRITZ STARLINGER (Vienna). Medium 8vo. Pp. 106 + vi. 1931. Vienna: Julius Springer. RM. 6.60.

*Die Indikationen zum abdominalen Kaiserschnitt für alle Kaiserschnittoperateure.* By Prof. Dr. GEORG WINTER (Königsberg). Royal 8vo. Pp. 124 + viii. 1931. Stuttgart: Ferdinand Enke. Paper covers, RM. 8; bound, RM. 9.60.

- The Rational Treatment of Varicose Veins and Varicocele.** By W. TURNER-WARWICK, M.A., M.B. (Cantab.), F.R.C.S., Surgeon to Outpatients and Surgeon in charge of the Rectal Department, the Middlesex Hospital, London. Crown 8vo. Pp. 188, with 12 illustrations. 1931. London: Faber & Faber. 5s. net.
- The Ætiology of Irregularity and Malocclusion of the Teeth.** Four lectures by Prof. J. C. BRASH, M.C., M.A., M.D. (University of Birmingham). With an Appendix "Concerning Orthodontic Problems and their Solution," by Miss M. L. TILDESLEY. Royal 8vo. Pp. 274 + xii, with 195 illustrations. 1931. London: Dental Board of the United Kingdom. 5s. net.
- Chirurgie de l'Ulcère gastrique et duodénal (Indications. Résultats).** By N. HORTOLMEI, Professeur de Clinique chirurgicale; and VL. BUTUREANU, Maître de Conférences, Faculté de Médecine de Jassy. Medium 8vo. Pp. 408, with 75 illustrations. 1931. Paris: Masson et Cie. Fr. 45.
- A Short History of Anatomy.** By RICHARD H. HUNTER, Ph.D., M.D., M.Ch., Lecturer in Anatomy, Queen's University, Belfast. Second edition. 1931. Crown 8vo. Pp. 88. 1931. London: John Bale, Sons & Danielsson Ltd. 3s. 6d. net.
- Surgical Pathology of the Diseases of Bones.** By ARTHUR E. HERTZLER, M.D., Professor of Surgery, University of Kansas. Hertzler's Monographs on Surgical Pathology. Royal 8vo. Pp. 272 + xiv, with 211 illustrations. 1930. London: J. B. Lippincott Co. 21s. net.
- Chirurgie du Rectum.** By HENRI HARTMANN, Professeur de Clinique chirurgicale, Chirurgien de l'Hôtel-Dieu. Imperial 8vo. Pp. 398, with 161 illustrations. 1931. Paris: Masson et Cie. Fr. 75.
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## *SOME BYGONE OPERATIONS IN SURGERY.*

BY SIR D'ARCY POWER, K.B.E., LONDON.

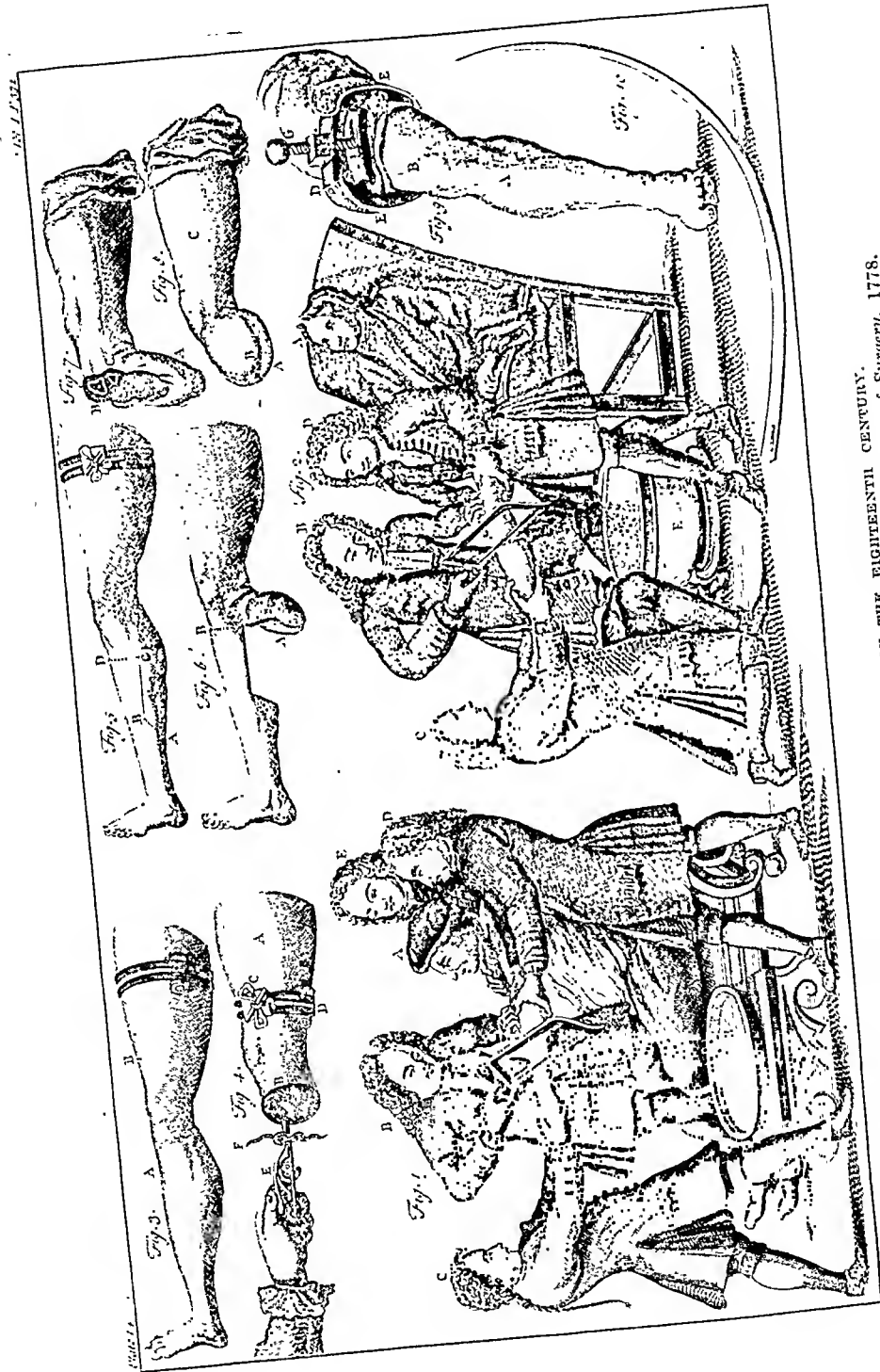
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### VII. AMPUTATION : THE OPERATION ON NELSON IN 1797.

(Concluded.)

EVERYONE knows that the pictures and portraits of Nelson represent him with his right sleeve empty and folded, but few remember the details of the injury which led to the amputation of his arm early in the morning of July 27, 1797, directly after the attack on Santa Cruz in Teneriffe, one of the Canary Islands. Attended by his stepson, Lieutenant Josiah Nesbit, Nelson was being rowed from the *Sea Horse* towards the mole of Santa Cruz on the night of July 24, 1797, when a grapeshot wounded him and seven of his boat's crew in their right arms. Nelson was in the act of drawing his sword and stepping out of the boat, but as he fell he grasped the sword in his left hand, for it had been given him by his uncle Maurice Suckling and he valued it highly. Lieutenant Nesbit, who was just behind him, heard the Admiral exclaim as he fell, "I am shot through the arm, I am a dead man." He immediately laid his stepfather at the bottom of the boat, and observing that the sight of the blood coming from the arm was making him faint took off his own hat to hide it from view. He then held the arm to stop the bleeding, examined the wound, and bound up the arm tightly with a handkerchief which he took off his own neck. Nelson said afterwards that he owed his life to this action. Nesbit was assisted by Lovel, one of the admiral's bargemen, who tore his shirt into strips to make a sling. Five other seamen were then collected, and with their help the boat was pushed afloat, for it had grounded with a receding tide. Nesbit took an oar and ordered the steersman to run close under the guns of the battery so that they might be safe from the tremendous gunfire.

The voice of his stepson giving the order roused Nelson from his faintness, and he immediately desired to be lifted up in the boat that, to use his own words, "he might look a little about him". He was accordingly raised by Nesbit, and Nelson saw that the *Sea Horse* would be the first ship the boat would reach. Nothing could induce the Admiral to go aboard it, though he was told that it might risk his life if they continued to row to another ship.



AMPUTATION AT THE CLOSE OF THE EIGHTEENTH CENTURY.  
From the English translation of Laurence Heister's *General System of Surgery*. 1778.

"Then I will die," he said, "for I would rather suffer death than alarm Mrs. Fremantle by her seeing me in this state when I can give her no tidings whatever of her husband." (Captain Fremantle was in command of the *Sea Horse* and Nelson had only recently attended his wedding.) The boat was accordingly rowed to the *Theseus*, where notwithstanding the increased pain and weakness Nelson peremptorily refused all assistance in getting up the ship's side, saying "Let me alone. I have yet my legs left and one arm. Tell the surgeon to make haste and get his instruments. I know I must lose my right arm, and the sooner it is off the better." Mr. Hoste (afterwards Captain Sir Henry Hoste), one of the midshipmen on the *Theseus*, wrote to his father: "At two o'clock in the morning Admiral Nelson returned on board, being dreadfully wounded in the right arm with a grapeshot. I leave you to judge of my situation when I beheld our boat approach with him who I may say has been a second father to me, his right arm dangling by his side, whilst with the other he helped himself to jump up the ship's side and, with a spirit which astonished everyone told the surgeon to get his instruments ready for he knew he must lose his arm and that the sooner it was off the better. He underwent the amputation with the same firmness and courage that always marks his character."

Whilst he waited for the necessary arrangements to be made for the operation Nelson busied himself with the relief party which he sent to rescue the survivors from the *Fox*, and then descended into the cockpit. By this time he was somewhat collapsed, for he said afterwards that the feeling of cold was severe, and he subsequently ordered that portable stoves should be supplied to the cockpit in every ship under his command.

The surgeon to the *Theseus* was Thomas Eshelby, who must have been quite a young man, for the records of the Surgeons' Company show that he was examined on April 7, 1791, and was found fit to be a third mate on a third-rate. He was examined again on Sept. 4, 1794, and was then reported as qualified for surgeon to a fourth-rate. He joined the *Theseus* by warrant on May 27, 1797, when he received a Bounty of £20. He took the place of Samuel Hardy, qualified to act as surgeon to a first-rate, who had been discharged to H.M.S. *Captain* on May 26, 1797. There is no mention of a second surgeon, but the Muster Roll contains an entry, "George Henderson 3rd. mate of Alloa, Scotland, joined by warrant 13th. March 1797 drowned at the storming of Santa Cruz." Eshelby, therefore, was short-handed at the time.

The amputation was performed by the circular method at the seat of election—that is to say, just below the insertion of the deltoid and at the point where the median nerve crosses the brachial artery. A good deal of force must have been used in retracting the soft parts, for it was realized later that the stump was unusually short. The subsequent progress appears in the extracts from the Medical Journal of H.M.S. *Theseus* reproduced on the next page.

Silk ligatures were used and the second ligature had included the median nerve as well as the artery. It proved to be a source of much trouble and pain, and did not come away until Dec. 4, 1797, when the sinus healed quickly. The pain which was accompanied by twitching of the stump was



Medical Journal of His Majesty's Ship the *THESEUS* . . . from the 26th May 1797 to the 18th August 1797 during which time the said Ship has been employed off Cadiz &c. &c. &c.

P. 7

May 27th 1797.

[Captain R. W. Miller  
Surgeon Thomas Eshelby]

MEN'S NAMES, AGES, AND QUALITIES	WHEN AND WHERE PUT ON THE SICK LIST	STATEMENT OF THE CASE WHEN PUT ON THE LIST	SYMPTOMS AND TREATMENT WHILE UNDER CURE	WHEN DISCHARGED TO DUTY, DIED, OR SENT TO THE HOSPITAL	REMARKS
Admiral Nelson P. 9	25 July	Compound fracture of the right Arm by a musket ball passing thro' a little above the Elbow; an Artery divided; the Arm was immediately Amputated, and the following give [sic] him R Opii gr. ij. f. Pil. statim s. Rep. Pil. Opii gr j— Rep. Pil. Opii gr ij hora s.s.			
Admiral Nelson P. 10	Page 7 <sup>th</sup>	Amputated Arm	Rested pretty well and quite easy. R Mist Salin. ʒij ter in die Rep <sup>r</sup> Pil opii hora s.s.		Tea, Soup & Sago, Lemonade & Tamarind Drink—
Admiral Nelson P. 11	Page 7 <sup>th</sup>	Amputated Arm	Had a middling Night. No fever. Rep <sup>r</sup> Pil Opii hora somni s.		
Admiral Nelson P. 12	Page 7 <sup>th</sup>	Amput <sup>d</sup> Arm	Dressed the stump, look'd well. R Decoet. Cinchon. ʒiss ter in die Rep Pil Opii h.s. s.		
Admiral Nelson P. 14	Page 7 <sup>th</sup>	Amputated Arm	Pretty easy. had no stool since the Operation. gave the follow <sup>s</sup> R Infus. Sentic ʒij Pulv. Jalap. Sp: Lav <sup>d</sup> co. ʒjs mane sum <sup>d</sup> Rep Pil. Opii hora somni s.		
Adm <sup>l</sup> Nelson P. 15	Page 7 <sup>th</sup>	Amputated	The Cathartic operated well. pretty easy. Rep. Pil Opii h.s. s. Rep. Decoet. Cinchon.		
Adm <sup>l</sup> Nelson P. 16	Page 7 <sup>th</sup>	Amputated Arm	One of the Ligatures came away. looks well. Rep. med. ut heri		
Admiral Nelson	Page 7 <sup>th</sup>	Amputated Arm	Continued getting well very fast stump look'd well. no bad symptom whatever occurred. he continued the use of the Cortex. and a gentle opening Draught occasionally.	Discharged* on board the <i>Sea-horse</i> 20th August * [sic]	The sore reduced to the size of a shilling, in perfect good health one of the Ligatures not come away.

so severe that Nelson was given opium from time to time for its relief. Elliot, afterwards Lord Minto, who saw him before the ligature came away, reports that he found Nelson looking better and fresher than he ever remembered him, although the continued pain prevented sleep. He was already impatient to go to sea again, and chafed under the delay of healing, concerning the duration of which the surgeons could give him no assurance. The ligature must be left to slough away, they said, for it was two inches up the wound, and if, attempting to cut it, the artery should be cut, another amputation higher up would be necessary, which would not be easy, for the stump was already very short. As soon as the ligature came away, Nelson, who had been lodging in Bond Street, sent a notice to the Rector of St. George's, Hanover Square, saying, "An officer desires to return thanks to Almighty God for his perfect recovery from a severe wound, and also for many mercies bestowed upon him."

The log of the *Theseus*, which is signed by Captain Ralph Willett Miller, merely says, "At  $\frac{1}{2}$  past 10 p.m. the seamen and marines left the ship under command of Rear Admiral Nelson. At  $\frac{1}{2}$  past 2 a.m. began a heavy cannonading in the Town. At  $\frac{1}{2}$  past 3 Admiral Nelson returned on board (being wounded in his right arm which occasioned it to be cut off.)"

I am indebted to the Secretary of the Admiralty and to Mr. Warren R. Dawson for several references. Miss E. H. Fairbrother kindly made the extract from the Medical Journal of H.M.S. *Theseus* which is printed on page 354. The document is preserved in the Public Record Office, Chancery Lane. The illustration taken from the 1778 English translation of Laurence Heister's *General System of Surgery* shows the method of amputation in use at the time of Nelson's operation.

## CONGENITAL LYMPHANGIECTATIC FIBROUS HYPERTROPHY (ELEPHANTIASIS CONGENITA FIBROSA LYMPHANGIECTATICA).

BY D. STEWART MIDDLETON, EDINBURGH,

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UNDER the heading of 'congenital elephantiasis' there have been described a very interesting series of congenital overgrowths. These are of considerable rarity, and it has been thought worth while to place on record a detailed report of such a case.

The term 'elephantiasis' has purposely been replaced by the word 'hypertrophy' in the title of this paper. 'Elephantiasis' has been used indiscriminately with reference to the filarial and non-filarial forms of enlargement, leprosy, congenital hypertrophy, congenital œdema, and to cases of local gigantism. The term 'elephantiasis' apparently originated as a soldier's slang expression in the Roman army during the Libyan campaign, but whether it referred to filarial elephantiasis, leprosy, or beri-beri will probably never be decided. However hallowed the expression may be by the lapse of time and by its passage through the pages of countless text-books, it is clearly neither descriptive nor associated with any scientific meaning. It should, therefore, be set aside for the delectation of the laity so far as the class of case dealt with in this paper is concerned.

### CASE REPORT.

A male child, age 10 months, was admitted to the Royal Edinburgh Hospital for Sick Children in November, 1928, and the following history was obtained :—

**HISTORY.**—Fourteen days before the infant was born the mother had a severe fall. The child was one of binovular twins, and the labour was premature by one month. Immediately after birth the child was noticed to have a swelling in the neighbourhood of the right wrist, and this was attributed by the accoucheur to an intra-uterine fracture of the distal extremity of the radius. Since birth, however, the right wrist steadily, though very slowly, increased in size, and the thickening extended from the wrist into the palm of the hand. The family history proved negative in so far as it was accessible.

**ON EXAMINATION.**—On examining the affected extremity a hard nodular swelling appeared to surround the distal end of the right radius. The skin was firmly bound to the underlying tumour, but the whole mass possessed a restricted degree of mobility upon the subjacent radius. The swelling extended into the palm of the hand, several hard nodules being present in the thenar and hypothenar eminences. Even at this stage the hand seemed to be completely functionless, and suggested a deformed hand which had been carved from a block of wood.

Radiographic examination showed no obvious erosion or enlargement of the radius, though the cortical bone on the lateral aspect of the distal half of the right radius was definitely sclerosed as compared with that on the left. The Wassermann reaction was negative.

On Nov. 13 a biopsy was performed. A portion of tissue was removed through an incision on the lateral aspect of the wrist. The tumour cut with the 'creaking' sensation typical of a hard fibrous structure, and the intimate connection between

the tumour and the overlying skin was very evident. A further small piece of tissue was removed from the hypothenar eminence for examination.

**PATHOLOGICAL REPORT.**—Dr. Agnes MacGregor, Pathologist to the Royal Edinburgh Hospital for Sick Children, reported upon these fragments of tissue as follows :—

“All parts of the mass examined are composed of dense mature fibrous tissue. When muscle is present at the edge of the mass, it is undergoing fibrous replacement, muscle fibres being separated by fibrous tissue and showing various stages of atrophy. In some such places the fibrous tissue is rather more cellular, but even then very well formed. The histological appearances are those of scar tissue rather than of neoplastic growth. If the condition is a tumour, the histological characters give no ground whatever for supposing it to be malignant.”

**PROGRESS.**—A short time later the child was demonstrated before a clinical meeting of the Edinburgh Medico-Chirurgical Society, and the consensus of opinion among those present was that, though it was a simple non-neoplastic condition at the moment, amputation was advisable in view of the likelihood of the occurrence of an ultimate fibrosarcomatous change.

The child was kept under observation for four months, and during this period the tumour gradually increased in size. The palm of the hand became more diffusely involved, the metacarpals being pushed apart and giving a trident-like appearance to the extremity. In addition an appreciable degree of extension took place proximally along the lateral aspect of the shaft of the radius.

At this time (March, 1929) it was decided to amputate. The reasons for coming to this conclusion were the steady increase in size of the tumour with its evident infiltrating character, the loss of prehensile power, and the ever-present fear of a malignant change, as frequently occurs in cases of diffuse angiomatous hypertrophy of a limb.

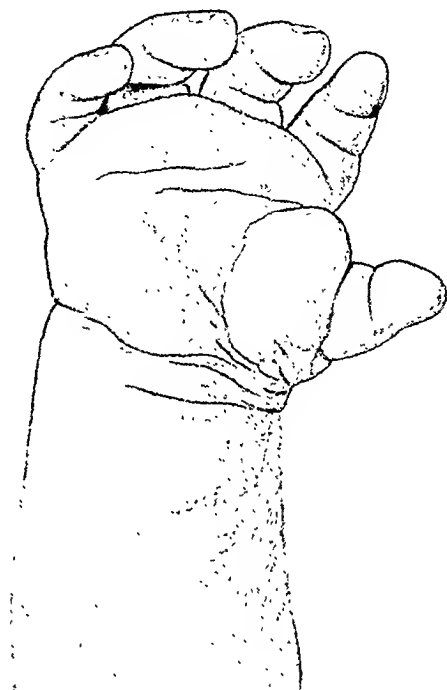


FIG. 269.—Drawing of the forearm and hand immediately after disarticulation. This shows the palmar overgrowth very well, though the portion surrounding the radius is not so clearly brought out.

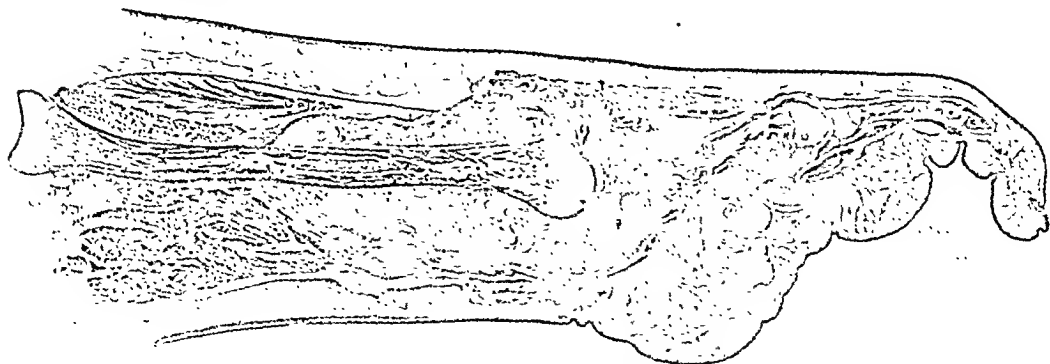


FIG. 270.—Longitudinal whole section of forearm and hand.

**OPERATION.**—On March 14, therefore, the forearm and hand were removed by disarticulation through the elbow-joint. The child has since remained well (May, 1931). *Fig. 269* shows the appearance of the hand at the time of removal.



**FIG. 271.**—Section to show the fibrous structure of the tissue with valved dilated lymph-vascular spaces.

fills the palm of the hand. The muscles of the forearm appear to be normal, but their tendons become incorporated in the fibrous mass at the wrist. Closer inspection shows the undisturbed fibres of the pronator quadratus muscle cut across at their insertion into the anterior aspect of the radius. In contrast to this, however, the small muscles of the hand have disappeared, and microscopical examination showed only the remains of a few fibres undergoing an apparent pressure atrophy.

More detailed microscopic examination shows the tumour to be composed of fully developed fibrous tissue. It is apparently simple scar tissue, and in no area does it demonstrate any neoplastic property. Scattered throughout this tissue and more especially situated below the skin are dilated lymph-vascular spaces which demonstrate very clearly the presence of fine valves (*Fig. 271*). No dilated blood-vascular spaces were seen. In many places the fibrous tissue extends up to the deeper layers of the dermis and is accompanied by a considerable degree of hyperkeratosis of the overlying stratum corneum (*Fig. 272*).

**HISTOLOGICAL EXAMINATION.**—Colonel Harvey, of the Laboratory, of the Royal College of Physicians of Edinburgh, undertook the complete histological demonstration of the specimen. The forearm and hand were frozen and sawn through in the long axis of the radius. A thin sheet of tissue was then sawn off one face of the specimen and decalcified. Large sections were taken from the whole surface and fitted together on mounting to show the whole length on one slide.

*Fig. 270* is a painting of this preparation as a whole section, done for me by Mr. J. Grieve. Inspection of the section shows that the mass of fibrous tissue has surrounded the distal half of the radius and



**FIG. 272.**—Section to show hyperkeratinization of the skin overlying the fibrous area.

extends up to the deeper layers of the dermis and is accompanied by a considerable degree of hyperkeratosis of the overlying stratum corneum (*Fig. 272*).

From the history and the clinical and histological examination, this appears to be a case of congenital fibrous lymphangiectatic hypertrophy affecting the fibrous connective tissues of the right wrist and hand.

### DISCUSSION.

I have found only one description of an apparently similar case. Spinner<sup>1</sup> describes a similar picture in which the congenital fibrous hypertrophy affected the right leg below the knee. Dilated lymph-vascular spaces were also present in large numbers, many being of macroscopic size. In his case, however, the condition was associated with an increase of growth in length of the bones of the leg—that is to say, a true gigantism.

Aschoff<sup>2</sup> divides congenital hypertrophies into :—

1. Local gigantism, in which there is merely an increase in bulk of a limb, all the tissues being equally affected, and which is unassociated with 'tumour' formation. Such a case affecting the right leg is shown in *Fig. 273*. Even in cases such as this, however, it is usual to see definite dilatation of the subcutaneous veins over a part of the overgrown limb. In the case figured definite venous dilatation was present in the thigh.

2. Congenital 'elephantiasis', which is not a uniform enlargement of all constituent parts of a limb with conservation of the normal proportions, but an overgrowth affecting only separate tissues and often leading to dreadful deformities.

According to the tissue involved Aschoff subdivides these enlargements into elephantiasis fibrosa, lipomatosa, angiomatosa, lymphangiomatosa, and neuromatosa. He also draws attention to the frequent occurrence of hyperkeratosis and warty proliferation of the skin overlying the hypertrophied area. Further, and most wisely, he safeguards this classification by stating that 'elephantiasis' and 'true local gigantism' may co-exist, just as we have seen them to do in Spinner's case.

It becomes quite obvious that hard-and-fast methods of classification cannot be applied to this class of deformity, as the following points will demonstrate :—

1. It is difficult, as I have already stated, to draw a sharp line of distinction between congenital fibrous hypertrophies of one kind or another and local gigantism, or even the hemi-hypertrophies affecting the whole of one-half of the body.

2. The majority of fibrous hypertrophies are associated with the presence of dilated lymph-vascular spaces in the fibrous element. It is of interest to note here that Spinner believes that the lymph-vascular dilatation is the



**FIG. 273.**—Female, age 5½. Shows local gigantism of the right leg. Considerable venous dilatation was present in this limb, but is not obvious in the photograph.

primary developmental fault and that the fibrosis is merely secondary to the resulting increase in nutrition of the part. The dilatation may be merely microscopic or the lymph spaces may form obvious cystic spaces. We are conversant with the ordinary infiltrating type of lymphangioma which is not uncommonly met with as a congenital tumour in the subcutaneous tissue of infants. Here we have numerous cystic lymph-vascular spaces embedded in a stroma of fibrous tissue. At what point can the line be drawn between the fibrous hypertrophy and the lymphangioma? A classification which ascribes a different pathogenesis to two similar processes according to the relative preponderance of the constituent parts of the tumour is likely to keep the truth for ever lost in a maze of words.

A case of fibrous hypertrophy may be associated with the presence of dilated blood-vascular spaces in the tumour, as in Aschoff's 'elephantiasis



FIG. 274.—Male, age 5½. A case of diffuse cavernous angioma of the right arm. The venous dilatation extends up to and ends sharply at the mid-line of the trunk both anteriorly and posteriorly.

congenita fibrosa angiomatosa'. If the fibrous element is relatively small, the condition receives the name of 'elephantiasis angiomatosa', as in the case depicted in *Fig. 274*, which is neither more nor less than a widespread cavernous angioma affecting the whole of the right arm and sharply delimited by the mid-line of the body anteriorly and posteriorly. It is clear that all intervening stages will be met with, and a classification which tries to separate the congenital enlargements of this type from the simple subcutaneous hæmangioma tends to make confusion worse confounded.

Enough has been said to show the futility of classification into closed compartments where no such compartments exist. One is more likely to

arrive at a true conception of the meaning of these difficult problems by discarding classifications and studying each case independently as a separate embryological problem. By such means we shall probably be able to link up this heterogeneous group of congenital overgrowths with many of the ordinary everyday congenital conditions met with in the out-patient department of a children's hospital.

I should like to acknowledge my indebtedness to Miss G. Herzfeld, F.R.C.S.E., for permission to deal with this case, and to Dr. T. Y. Findlay and Mr. Norman Dott for permission to utilize *Figs. 273 and 274*.

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## TWO CASES OF LOCALIZED PHLEGMONOUS ENTERITIS.

By C. GORDON IRWIN AND STUART McDONALD, JUNR.,  
NEWCASTLE-UPON-TYNE.

ACUTE localized phlegmonous enteritis involving a small circumscribed area of bowel is a condition which occurs with such rarity that a description of the clinical history, symptoms, and pathological findings in the two cases to be described is worthy of record.

### LITERATURE.

Though phlegmonous inflammation of the submucosa of the intestine was described by Rokitsansky<sup>1</sup> as early as 1842 there have been remarkably few cases recorded, and as regards British literature we have been unable to find records of a case similar in distribution to our own two examples.

Belfrage and Hedenius<sup>2</sup> record a case in which a man of 52, whose previous health had been good, was admitted to hospital suffering from violent abdominal pain and vomiting and absolute constipation. He died three and a half days after the onset, and at the autopsy a phlegmonous enteritis extending for 18 cm. below the commencement of the jejunum was found. There was an associated purulent peritonitis.

Eskridge<sup>3</sup> reports a case which occurred in a woman of 55 who had suffered from attacks of vomiting and indigestion for fourteen years. The onset of the final illness was sudden, with abdominal pain, vomiting, and a rigor, and death occurred after ten days. She had had a similar attack two months previously. At the autopsy a phlegmonous duodenitis with gangrene of the mucosa was found extending for 12 in. from the pylorus. There was a large stone in the gall-bladder, but this was apparently unconnected with the condition.

Weichselbaum<sup>4</sup> gives a post-mortem report of a case of phlegmonous duodenitis, in which there was great thickening of the bowel wall, with scattered small purulent collections, but no ulceration. There are no clinical notes.

Askanazy<sup>5</sup> reports a case in which a man of 51 was admitted to hospital suffering from severe abdominal pain and died in two days. Fourteen days previous to admission he had fallen from a beam and injured his knee, and since then complained of pains in the trunk. At the autopsy there was serous peritonitis with phlegmonous inflammation of the transverse part of the duodenum. Microscopical examination showed numerous cocci in the bowel wall, and a culture of streptococci and *S. albus* was obtained.

Moisejev<sup>6</sup> records a case in which a man of 67 who had been ill for an unknown length of time was admitted suffering from dyspnoea with pain in the side and acute pleurisy. He died twenty-four hours after admission. At

the autopsy there was phlegmonous inflammation of the first 40 cm. of jejunum, with fibrino-purulent peritonitis. Microscopical examination showed the presence of streptococci.

Matthes<sup>7</sup> describes a case (female of 53) which was fatal from cirrhosis of the liver with ascites, complicated by an ulcer of the leg with surrounding erysipelas. At the autopsy it was found that the entire small and large intestine was the seat of phlegmonous inflammation, the maximum intensity being in the first 32 cm. of the small intestine below the ampulla of Vater. There was an associated fibrino-purulent peritonitis, with acute lymphadenitis of the mesenteric and retroperitoneal glands, and infiltration of the mesentery with seropus. A pure culture of streptococci was obtained.

Deutelmoser<sup>8</sup> reports a case in which a man of 21 was admitted to hospital complaining of jaundice, vomiting, and abdominal pain and tenderness. Symptoms had commenced fourteen days previously. The patient died forty days after onset, and at autopsy a phlegmonous inflammation of the duodenum was found, this being maximal in the neighbourhood of the ampulla of Vater. The head of the pancreas was œdematous and the surrounding lymph nodes were inflamed, but there was no peritonitis. Microscopical examination of the bowel showed large numbers of streptococci.

MacCallum<sup>9</sup> describes a case in which a man of 75 was admitted to hospital complaining of violent pain in the upper part of the abdomen and incessant vomiting of bile-stained material. He had been run over by a wagon three weeks previously and had suffered head injuries; he complained of abdominal pain at this time. The patient died two days after laparotomy, and at the autopsy it was found that a phlegmonous enteritis extended for 5 cm. below the pylorus for a distance of 30 cm. Localized peritonitis was present. Microscopically, myriads of streptococci were demonstrated in the bowel.

Ungermann<sup>10</sup> describes a case in which a man of 60 was admitted to hospital with a history of violent abdominal pain and vomiting of sudden onset, together with jaundice. The patient died in coma after three days. Autopsy showed a phlegmonous inflammation of the duodenum and adjacent parts of pylorus and duodenojejunal junction; this was most marked in the descending part of the duodenum. Close to the ampulla of Vater there was a diverticulum, the walls of which were the seat of a dense purulent inflammatory process. Lying free in the floor of the diverticulum were some sharp splinters of bone. There was marked œdema of the tissues surrounding the duodenum and a diffuse purulent peritonitis was present. The liver showed a portal infiltration with pus cells. On microscopical examination of the bowel large numbers of streptococci were found. This was most marked round the ampulla of Vater.

Ingier<sup>11</sup> states that localized phlegmonous enteritis is a rare complication of lobar pneumonia, and quotes a case in which a man who died from lobar pneumonia had a localized phlegmon of the cæcum and ascending colon, 12 cm. in length. Pneumococci were demonstrated in the bowel wall.

Taylor and Laking<sup>12</sup> describe a case in which a woman of 66 was admitted to hospital suffering from abdominal pain and repeated vomiting of thirty-six hours' duration. Laparotomy was performed, and infiltration of the duodenal

wall noted. Death occurred thirty-six hours after operation. At the autopsy there was found a phlegmonous enteritis of the second part of the duodenum. In the midst of the affected area there was a fish-bone partly buried in the mucosa. A pure culture of *B. coli* was obtained from the duodenal wall and also from an effusion at the base of the right lung.

Dowd<sup>13</sup> reports a case in which a man of 23, who had suffered from intermittent pain in the left side of the abdomen for one year, was admitted to hospital complaining of acute abdominal pain and passage of blood per rectum. The onset was two and a half days previously. At laparotomy the whole of the descending colon was found to be the seat of a purulent inflammation, and was resected. The patient made a good recovery. On microscopical examination of the colon a phlegmonous inflammation with Gram-positive cocci was found.

Frising and Sjövall<sup>14</sup> quote two cases. In the first, a woman of 57 was admitted suffering from violent abdominal pain, bile-stained vomiting, and shivering of twelve hours' duration. Previous health was good. At the autopsy a phlegmonous inflammation of the duodenum and a few centimetres of jejunum was found, associated with œdema of the head of the pancreas and purulent infiltration of the retroduodenal tissues. Part of a fish-bone was found in the duodenal contents. On microscopical examination numerous streptococci were found in the bowel wall.

In the second case, a female lunatic of 73 was admitted suffering from severe abdominal pain, bilious vomiting, and shivering. She died the day after admission. At the autopsy a phlegmonous inflammation of the duodenum and 1 cm. of jejunum was found. This was most marked in the descending part of the duodenum. A diverticulum was present, close to the ampulla of Vater. There was purulent infiltration of the head of the pancreas and the retroduodenal tissues. There was no peritonitis. On microscopical examination of the bowel wall abundant streptococci were found.

Müller<sup>15</sup> describes a case in which a man of 51, whose previous health had been good, was admitted to hospital suffering from violent abdominal pain of sudden onset. At operation the appendix was slightly inflamed and was removed. There was abundant turbid effusion in the pelvis. The patient died the following day. At the autopsy some perityphilitis was present and there was acute generalized peritonitis. A phlegmonous inflammation was present in a high coil of the jejunum and there was a large abscess between the leaves of the mesentery. Streptococci were demonstrated in the affected jejunum and in the mesentery.

Von Saar<sup>16</sup> reports two cases. In the first a man of 21 was admitted to hospital complaining of severe pain in the right side of the abdomen, vomiting, and absolute constipation. He had suffered from epigastric pain after food for some years previously. At operation there was found acute generalized peritonitis with much turbid effusion. The whole of the ascending colon was the seat of phlegmonous inflammation. It was brought out and fixed in the abdominal wound, and was later removed with a Paquelin's cautery. The patient made a good recovery, and continuity of the bowel was restored in five weeks.

In the second case, a man of 44 was admitted to hospital with a five days' history of constipation, colicky pains in the abdomen, and vomiting foul-smelling material. His previous health was good. At operation a high coil of jejunum about 13 in. long was found to be the seat of phlegmonous inflammation. There was an associated patchy fibrinous peritonitis on the parietal peritoneum. The mesenteric glands were inflamed. The affected coil of bowel was packed off, and the patient died three days later. At autopsy the diagnosis of primary phlegmonous jejunitis was confirmed.

Black<sup>17</sup> reports two cases. In the first a woman of 53, previously healthy, was admitted suffering from severe upper abdominal pain and persistent vomiting. She had had a rigor. At operation twenty-four hours after onset, plastic peritonitis round the duodenal area was noted. Drainage was employed, but the patient died shortly afterwards. At the autopsy phlegmonous inflammation of 8 in. of duodenum was found, starting 2½ in. from the pylorus. Cultures from the liver, spleen, and duodenal wall grew streptococci.

In the second case, a man of 55 was admitted suffering from abdominal pain and vomiting followed by a rigor. He had had attacks of abdominal pain and vomiting for years. The vomiting continued, and there was a second rigor thirty-six hours after the onset. At operation the whole of the duodenum and 8 in. of the jejunum were found to be the seat of phlegmonous inflammation. A posterior gastrojejunostomy was performed, but the patient died thirty-two hours after operation.

Glaus<sup>18</sup> describes a case in which a man of 53 was admitted to hospital suffering from some (?) pulmonary condition with dry cough. Beyond some meteorism there was no evidence of abdominal disease. The patient died after a short illness. At the autopsy chronic pulmonary tuberculosis of cavernous nodose type was found, with tuberculous disease of the peritoneum, suprarenals, liver, and spleen. Gall-stones were present. In the lower part of the ileum there was a localized phlegmonous enteritis. The spleen showed acute splenic tumour, and a culture of *S. pyogenes aureus* was obtained. The wall of ileum showed purulent inflammation with Gram-positive cocci and Gram-positive bacilli. The interest of this case lies in the clinical latency of the condition.

Hellström<sup>19</sup> gives a full account of phlegmonous enteritis, and quotes five cases. In the first case a man of 42, previously healthy, was admitted with a three days' history of abdominal pain which increased in severity, vomiting of foul-smelling material, and finally absolute constipation. He had a rigor at the onset. At operation phlegmonous inflammation was present in a high coil of jejunum, together with localized peritonitis. The affected coil was isolated and jejunostomy performed. The patient died shortly afterwards, and at autopsy it was found that the first 30 cm. of jejunum was affected.

In the second case, a man of 55, previously healthy, was admitted complaining of violent abdominal pain. There was tenderness on rectal examination. At operation purulent peritonitis was present. Drainage was employed, but the patient died three days later. At the autopsy a portion of jejunum 10 cm. in length, and commencing 30 cm. below the duodenojejunal flexure,

was found to be the seat of phlegmonous inflammation with associated sero-fibrinous peritonitis. The related mesentery was thickened and the lymph nodes were inflamed.

The third case occurred in a woman of 54, previously healthy save for an operation for gangrenous appendicitis thirteen years previously, this being followed by a ventral hernia. She was admitted suffering from violent abdominal pain and bilious vomiting, and this was followed by a rigor. At operation twenty-four hours after onset turbid effusion was present in the peritoneum, and a fistula was found between the duodenum and gall-bladder. The latter was removed and the duodenum sutured. As the lumen of duodenum was so much narrowed a gastro-enterostomy was performed, but a portion of jejunum 30 cm. long was inflamed and thickened and had to be resected. The patient died thirty-nine days later. At the autopsy a portion of jejunum 60 cm. below the duodenojejunal flexure was the seat of phlegmonous inflammation, and there was an abscess between the leaves of the related mesentery.

The fourth case was that of a man of 37 who had had symptoms suggestive of a gastric ulcer for one year. He had also complained of bloody stools and tenesmus during this time. Twenty-four hours before admission he collapsed with severe pain in the left lumbar region and vomiting. The bowels were loose. Laparotomy was performed, but no free fluid was found. Complete anuria occurred and renal decapsulation was performed on the next day. At this operation it was found that the whole of the descending and pelvic colon, especially at the sigmoid flexure, was swollen; the wall of the bowel being bluish-red in colour. The patient died the next day. At the autopsy the peritoneal cavity was found to contain clear serous fluid. The colon, from the left colic flexure to the rectum, was the seat of phlegmonous inflammation. The mesocolon was swollen and the retrocolic tissues were œdematous. The kidneys showed acute nephritis. On microscopic examination there was purulent inflammation of the wall of the colon, and numerous streptococci together with Gram-positive putrefactive organisms were present.

The fifth case occurred in a man of 46 who had had digestive troubles for many years. His symptoms had become worse fourteen days prior to admission. He complained of violent abdominal pain and vomiting. At the operation an opalescent exudate was present below the transverse colon and it was found that 30 cm. of a high coil of jejunum was swollen and inflamed. The inflammatory process commenced 40 cm. below the duodenojejunal junction. The affected loop was excised and lateral anastomosis effected. The patient died two days later, and at the autopsy the diagnosis of phlegmonous jejunitis was confirmed. The mesentery was thickened. Streptococci were demonstrated in the bowel wall.

Adams<sup>20</sup> reports one case which occurred in a man of 68 who had had dyspepsia of some months' duration. He was admitted to hospital with a seven days' history of intermittent epigastric pain, vomiting, and passage of offensive blood-stained stools. At operation it was noted that most of the coils of small intestine were gangrenous. At autopsy most of the small and large bowel was swollen and œdematous. There was no mesenteric thrombosis.

Bohmansson<sup>21</sup> in reviewing the Continental literature records 68 cases of acute purulent inflammatory processes in the submucosa and subserosa of the intestines, including 11 cases collected by himself.

Analysis of the recorded cases as regards the site of the lesion gives the following figures :—

	No. of Cases	Per cent
Entire gastro-intestinal tract ..	1	1·5
Duodenum .. ..	18	26·5
Free part of small intestine ..	19	28·0
Colon .. ..	30	44·0

The distribution of Bohmansson's own cases was as follows :—

Ileum .. ..	6
Cæcum .. ..	1
Descending colon ..	1
Duodenum .. ..	3

On referring to the above 11 cases, it would appear that only in some of them were the clinical and pathological investigations sufficiently thorough to allow of a definite diagnosis of phlegmonous enteritis being made.

### CASE RECORDS.

*Case 1.*—The patient, a somewhat obese male, age 51, a foy boatman by occupation, was admitted to the Royal Victoria Infirmary at 4 p.m. on Jan. 17, 1931.

**HISTORY.**—Up till four days before admission he had led an active and healthy life and gave no history of previous disease. His first symptom was pain of sudden onset, localized to the epigastrium and accompanied by marked shivering and later sweating. The pain was intermittent and recurred in the vicinity of and immediately above the umbilicus. A few hours after the onset vomiting began and continued with increasing severity up till his removal to hospital. The bowels were constipated and there had been no movement for three days, and for two days he had not passed flatus.

**ON EXAMINATION.**—The patient was cold and collapsed, with a feeble thready pulse of 120 and a temperature of 100°. The tongue was dry and thickly coated, and he was vomiting foul-smelling material at intervals. The abdomen was distended and tender throughout, with maximum pain in the umbilical region. There was no visible peristalsis. The hernial sites were normal, and rectal examination showed nothing abnormal. There were no indications of cardiovascular or of respiratory disease. A tentative diagnosis of mesenteric thrombosis was made and immediate operation carried out.

**OPERATION.**—A mid-line incision above the umbilicus was employed. On opening the peritoneum marked suppurative peritonitis with much effusion was found. The omentum was found to be firmly adherent to a loop of small bowel. This was freed, and about 9 in. of bowel was seen to be œdematous, enormously thickened, acutely inflamed, and covered with patches of adherent lymph. The vessels were inspected and found to be pulsating normally. The bowel on either side of the affected area was somewhat distended. A lateral anastomosis between loops immediately above and below the thickened area was carried out and tube drainage employed. The patient's general condition was bad, and he did not rally after the operation, dying twenty hours later.

### PATHOLOGICAL INVESTIGATION.

A post-mortem examination (No. 23/31) was performed on Jan. 19, 1931. The following report includes only relevant matter :—

**Peritoneal Cavity.**—Marked acute generalized peritonitis was present, especially in the upper part of the cavity. There was considerable turbid effusion.

The great omentum and coils of small intestine were pasted together with inflammatory exudate. Twelve inches below the duodenojejunal junction was a dilated, œdematous, and congested portion of jejunum which had been isolated by lateral anastomosis. This was removed intact. The bowel above the anastomosis was dilated, but below the anastomosis it was almost empty in parts. There was no gross abnormality in the large intestine. The stomach was markedly dilated and contained foul-smelling material. Beyond numerous petechial hæmorrhages beneath the mucosa there was nothing else abnormal in the stomach or duodenum.

The spleen, liver, kidneys, suprarenal glands, and pancreas showed no special gross changes other than those attributable to toxæmia. The urinary bladder and prostate gland showed nothing abnormal. There was nothing abnormal in the aorta or inferior vena cava or in their branches.

**Thoracic Cavity.**—There was no evidence of pleurisy or pericarditis. The heart showed no abnormality beyond some dilatation of the right auricle, atheroma of the first part of the aorta and coronary arteries, and toxic changes in the myocardium. There was nothing abnormal in the main bronchi, pulmonary artery, or lymph nodes at the root of the lungs. The lungs on section showed marked terminal passive congestion and œdema at the base on both sides. There was no evidence of pneumonia or tuberculosis.

**Portion of Jejunum removed at Post-mortem.**—

**ANATOMICAL DESCRIPTION.**—The affected loop of jejunum was 10 in. long. It was distended with fluid and appeared to be œdematous. There was evidence of



FIG. 275.—Case 1. Portion of jejunum removed at post-mortem.

early acute peritonitis. It was pale in colour, but the superficial vessels were congested and there were numerous sub-peritoneal petechial hæmorrhages. This was most marked on the lower portion of the loop. Throughout the affected portion of bowel many small yellowish areas could be seen shining through the peritoneal coat. The bowel above and below the affected loop showed no special change save some dilatation of the superficial vessels. On dissection of the main vessels supplying the affected loop no evidence of embolism or thrombosis could be found. There was no special change in the mesentery of the affected loop. On opening the bowel a large amount of thick yellowish fluid escaped which had a faint mawkish odour. A 'window' was cut in the anterior aspect of the loop so as to expose the interior of the bowel (Fig. 275). There was no evidence of the presence of

a foreign body. In the central portion of the specimen for an extent of  $7\frac{1}{2}$  in. the bowel wall was found to be greatly thickened, varying from  $\frac{1}{8}$  in. to  $\frac{1}{2}$  in. in places, the measurement being taken from the peritoneal coat to the tips of the valvulæ conniventes (Fig. 276). The remainder of the bowel at each end of the specimen showed no gross abnormality. The thickening was mainly at the expense

of the submucous tissues and mucous membrane, but the peritoneal and museular coats were also abnormally thick and were dark in colour, being in strong contrast to the submucosa and mucosa, which were intensely pale and had a vitreous appearance in some places. The normal mucosal folds were exaggerated,



FIG. 276.—Case 1. Longitudinal section through wall of jejunum. ( $\times 3$ .)

and in one place situated at the upper portion of the loop the mucosa was thrown up into folds so as to suggest a polypoid growth. The superficial layers of the mucosa showed extensive desquamation with hæmorrhage, and were necrotic in parts, the appearances indicating an intense inflammatory process, phlegmonous in type.

**HISTOLOGICAL DESCRIPTION.**—Sections were stained with hæmatin and eosin, and hæmatin and van Gieson.

1. *Mucous Membrane.*—This was thrown up into regular folds, and there was no evidence of neoplasm either simple or malignant, or of a granulomatous condition. Under the low power the villi were seen to be swollen and elongated and to be more cellular than normal. Inflammatory œdema was marked, especially in relation to the acini of the glands. There was extensive desquamation of the elements of the mucosa, and in some places there was superficial ulceration, the villi being destroyed and being replaced by a hæmorrhagic inflammatory exudate (Fig. 277). The connective tissue separating the mucosa and submucosa was, however, intact. Under the high power the mucosa showed degenerative changes which varied between intense cloudy swelling and nuclear degeneration and complete necrosis. The blood-vessels of the villi were congested and showed proliferation of the reticulo-endothelial cells lining their walls, and in the stroma of the villi was a cellular and fibrinous inflammatory exudate in which both polymorphs and mononuclear macrophages were abundant.



FIG. 277.—Case 1. Mucosa of jejunum showing ulceration. ( $\times 70$ .)



2. *Submucous Tissue*.—Under the low power the submucous connective tissue could be clearly made out, but was congested and infiltrated with a cellular and fibrinous exudate similar to that described above. The remainder of the submucous tissue was represented by a cellular mass extending in a broad band up to the muscular coats of the bowel (*Fig. 278*). Threads of fibrin were interspersed throughout the mass, and in some places the exudate was purely fibrinous. Extensive inflammatory oedema was present, and numerous congested blood-vessels could be seen. Under the high power the cellular constituents of the submucous exudate were found to consist of numerous polymorphs and mononuclear macrophages, the former predominating.

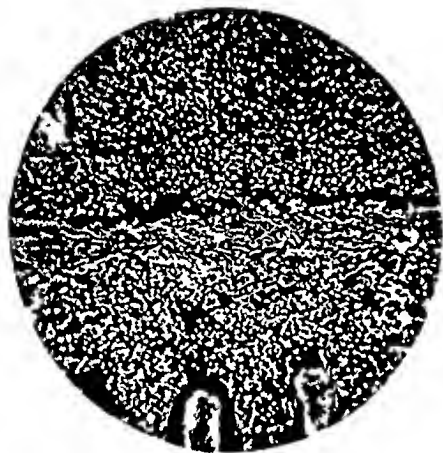


FIG. 278.—Case 1. Mucosa and submucosa of jejunum showing inflammatory exudate. ( $\times 210$ .)



FIG. 279.—Case 1. Peritoneal and longitudinal muscle coats of jejunum showing inflammatory exudate. ( $\times 210$ .)

3. *Muscular and Peritoneal Coats*.—Under the low power the muscle bundles of the circular coat were widely separated by extension of the inflammatory process from the submucosa, and similar appearances were seen in the longitudinal coats. The peritoneal coat was also the site of a cellular invasion, was intensely congested, and showed superficial erosions. Under high power the muscle fibres of the circular and longitudinal coats showed intense cloudy swelling and degenerative nuclear changes. The serosa cells of the peritoneal coat were swollen and disintegrated in many places. The character of the cellular exudate was similar to that described above (*Fig. 279*).



FIG. 280.—Case 1. Exudate in submucosa showing inflammatory cells and streptococci. ( $\times 645$ .)

**BACTERIOLOGICAL EXAMINATION.**—Stains used were carbol thionin, Gram, and Gram-Weigert.

*Smears from Bowel Contents.*—These showed numerous streptococci in short-chained and diplococcal forms, together with Gram-positive and Gram-negative bacilli. Many degenerate leucocytes were present.

*Smears from Surface of Mucosa.*—These showed similar appearances, but the streptococci were relatively more numerous.

*Section of Bowel Wall.*—In relation to the mucosa comparatively few organisms were present, these being mainly Gram-positive bacilli. On passing towards the submucosa enormous numbers of streptococci in diplococcal and short-chained forms were seen (*Fig. 280*). Phagocytosis was active. Bacilli were practically absent in the submucosa. Organisms were very scanty in the muscular and peritoneal coats, but were seen in relation to the cellular exudate which was separating the muscular bundles.

Cultural examination was not undertaken.

*Case 2.*—We are indebted to Professor G. Grey Turner for access to his notes of this case and to the specimen which he has preserved.

*HISTORY.*—The patient, a male, age 65, a farmer by occupation, was admitted to Professor Turner's private hospital on Jan. 25, 1913, with the following history. He had suffered from a large left serotal hernia of very long standing. Forty-eight hours before admission he had an attack of severe abdominal pain associated with shivering.

*ON EXAMINATION.*—The abdomen was very tender all over, but was not distended. The pulse was 100 and the temperature  $101.8^{\circ}$ . The patient's condition rapidly became worse and he was admitted with a provisional diagnosis of strangulated hernia. On admission he looked like a man dying of peritonitis.

*OPERATION.*—The hernial sac was found to contain a large amount of small intestine which was somewhat congested and inflamed. There was no actual strangulation, and it did not appear that the severe symptoms could be explained from the state of the hernia. The incision was extended and an affected loop of bowel was found high up on the left side of the abdomen. The appearance of the intestine resembled that of a gangrenous appendix, being greatly swollen, very much thickened, and with yellowish points all over its surface. The conclusion at the operation was that the loop of intestine had been inside the hernial sac and had not recovered after its return to the abdomen. As the patient was extremely ill the loop was surrounded with tissue drains and the abdomen closed. He became steadily worse and died about twelve hours later.

#### PATHOLOGICAL INVESTIGATION.

At the autopsy the loop of bowel was found to be in much the same condition as at the time of the operation. It proved to be a part of the upper jejunum and could not by any chance have been in the hernial sac. Beyond some little peritonitis the rest of the abdominal contents were healthy. The notes state that the heart was diseased, but the precise lesion was not mentioned.

##### Portion of Jejunum Removed at Post-mortem.—

*ANATOMICAL DESCRIPTION.*—The specimen consisted of a portion of jejunum 14 in. long, together with the mesentery in relation to it (*Fig. 281*). A 'window' had been cut in the bowel so as to expose its interior. The central portion of the specimen,  $5\frac{1}{2}$  in. long, showed great thickening of the bowel wall varying from  $\frac{3}{8}$  in. to  $\frac{5}{8}$  in. in places, the measurement being taken from the peritoneal coat to the tips of the valvulae conniventes. The remainder of the bowel at each end of the specimen showed no gross pathological change. In the affected portion the valvulae conniventes were thrown up into polypoidal masses, and the thickening was mainly confined to the mucosa and submucosa, though the muscular and peritoneal coats were also abnormally thick. The submucosa was yellowish-white in colour but in some places was discoloured from altered blood. The surface of the mucosa was the seat of intense inflammatory change and was completely necrotic in places, and there was evidence of considerable superficial hæmorrhage.

Examination of the external aspect of the entire specimen showed the presence of diffuse purulent peritonitis, most marked over the thickened portion. In the latter area numerous extensive purulent deposits could be seen shining through the peritoneum. The distribution of these deposits suggested the presence of a purulent

lymphangitis spreading towards the mesenteric aspect of the bowel. In this area there was a mass of acutely inflamed lymph nodes. Numerous small subperitoneal hæmorrhages were present, especially at the antimesenteric border of the thickened portion. Dissection of the main vessels supplying the bowel failed to reveal any evidence of embolism or thrombosis, nor did the colour of the bowel suggest that vascular obstruction had been the cause of the condition.

The gross appearances of the specimen suggested a phlegmonous inflammatory process.

**HISTOLOGICAL DESCRIPTION.**—Sections were stained with hæmatoxylin and eosin, and hæmatoxylin and van Gieson. Notwithstanding the fact that the specimen had been preserved in glycerin for eighteen years before sections were cut, there was remarkably little obscuration of fine histological detail.



FIG. 281.—Case 2. Portion of jejunum removed at post-mortem.

**Bowel Wall.**—In general the appearances corresponded exactly to those already described under *Case 1*, but the inflammatory process appeared to be more intense. The mucosa showed acute inflammatory change throughout, and in many places the appearance varied between superficial desquamation and ulceration, and complete necrosis. There was no evidence of neoplasm or of a granulomatous condition. The submucous connective tissue was clearly defined, but extending from the mucosa and infiltrating the circular and longitudinal muscle layers was an inflammatory exudate, consisting of polymorphs and mononuclear macrophages interspersed with threads of fibrin. The peritoneal coat showed intense acute inflammatory change with superficial erosions.

Under the high power numerous minute yellowish-brown granules were seen, both in mucosa and submucosa. The appearances suggested the precipitations so frequently seen in old formalin specimens. A positive prussian-blue reaction in some of the granules indicated that hæmosiderin was present, but on treating sections with caustic potash the majority of the granules disappeared.

**Section of Mesentery.**—This included the main vessels supplying the affected portion of the bowel and also an enlarged lymph node. The main vessels showed no evidence of thrombosis, but the artery was the seat of a marked degree of arteriosclerosis. The lymph node showed acute lymphadenitis with marked reticulo-endothelial hyperplasia and infiltration with polymorphonuclear leucocytes.

**BACTERIOLOGICAL EXAMINATION.**—Sections were stained with carbol thionin, Gram, and Gram-Weigert.

*Section of Bowel Wall.*—In relation to the mucosa large numbers of organisms were present, these being mainly Gram-positive bacilli of various types and short-chained streptococci. On passing towards the submucosa the appearances in some places corresponded to a pure culture of streptococci in short-chained and diplococcal forms; few bacilli were present. Phagocytosis was active. Streptococci were abundant in the exudate infiltrating the muscular coats of the bowel, but were scanty in the peritoneal coat.

*Mesenteric Lymph Node.*—No organisms could be demonstrated.

### SUMMARY.

1. Two cases of acute localized phlegmonous enteritis of streptococcal origin are reported.
2. In common with many of the cases recorded there was a sudden onset associated with a rigor and acute abdominal symptoms suggestive of acute intestinal obstruction.
3. There was a rapidly fatal termination with peritonitis.
4. Vascular obstruction and foreign body were excluded as etiological factors, and there were no indications of pyæmic deposits elsewhere in the body.
5. Pathological investigation suggests an autogenous infection possibly resulting from local injury of the bowel wall.

We are indebted to Mr. Trevor Barnett and to Mr. A. R. D. Pattison for valuable assistance in reviewing the literature relative to phlegmonous enteritis.

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**SPONDYLOLISTHESIS.\***

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DISPLACEMENT of the presacral spine forwards upon the sacrum has attracted a great deal of attention since the name of spondylolisthesis was applied to it by Kilian in 1854. In the latter half of the nineteenth century those chiefly interested were the obstetricians, who, stimulated by the work of Neugebauer, recognized the lesion as an occasional cause of difficult labour. With the advent of roentgenology fresh interest was aroused, and it has become apparent that this condition is at least as common in men as it is in women. A further development has been provided by improved lateral roentgenography of the spine, which has made possible the detection of lesions not seen in antero-posterior views. At the present time spinal lesions are being increasingly investigated because of the onslaught of the motor-car and of other mechanical devices. In view of its possible relation to trauma, the subject of spondylolisthesis occasionally provides material of medico-legal interest.

The present paper is based upon a study of 34 cases of spondylolisthesis, and of anatomical and roentgenological material, with special reference to the mechanics of the lesion and to its diagnosis. The main features of interest are summarized in the following table :—

	Cases
Males (53 per cent) .. .. .	18
Females (47 per cent) .. .. .	16
Under 25 years of age (youngest 11) .. .. .	13
Between 25 and 50 .. .. .	15
Over 50 years of age (oldest 71) .. .. .	6
Spondylolysis with displacement .. .. .	23
Forward displacement of entire 5th lumbar vertebra .. .. .	6
Displacement of 5th, but of uncertain type .. .. .	5
Early cases—mild displacements .. .. .	9
Spina bifida present .. .. .	15
Sciatica prominent symptom .. .. .	12
Severe injury to spine .. .. .	3
Moderate injury to spine .. .. .	6

**Types.**—There are two types of spondylolisthesis. The first is one that is often described in text-books and occasionally in journal articles, but is rare. In this practically the entire 5th lumbar vertebra slips forwards upon the sacrum and carries the rest of the spine with it. The second type, the one which is most frequently seen, and which was well recognized by Neugebauer as well as by most modern writers, consists in the separation (or spondylolysis) of the 5th lumbar vertebra into two portions by a solution of bony continuity in the laminae posterior to the transverse processes, in such a way

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that the part bearing the spinous process and inferior articular surfaces moves backwards and the rest of the vertebra slips forwards upon the sacrum. This is the condition discussed in this article.

**Literature.**—There have been written, up to the present time, about 250 articles upon this subject, a considerable number being of German or American origin. Until 1900 almost all of them were based upon descriptions of museum specimens, the pioneer work being carried out by Neugebauer. In England, Lane in 1885 discussed spondylolisthesis as one of the numerous changes found in the spine of labourers; but one of the best descriptions of pathological material was by Lawrence in 1900. The remarks by Blacker supplemental to Lawrence's paper are of importance, as he clearly appreciated that the site of bony defect was in the interarticular portion of the laminae of the 5th lumbar vertebra. Of modern British clinical and roentgenological work, that by Brailsford is the most noteworthy. In America in 1912, following a description of a case, Fitch gave an important list of 161 references to the literature. In recent years Whitman, Albee, Kleinberg, and Willis have made valuable contributions. An article by Meyerding in 1931 reporting 121 cases is of great interest from the clinical point of view. In this series 62 per cent of the patients were males. In 1928 Faldini in Italy made an excellent contribution to the study of spondylolisthesis. He applied a different title to each of the two types of lesion: for the rare slip of the entire 5th lumbar vertebra he reserved the name 'spondylolisthesis'; for the commoner lesion he used the title 'spondylolysis'.

**The Congenital Nature of the Basic Lesion of Spondylolisthesis.**—Many writers have noted the congenital nature of the defect in the laminae of the last lumbar vertebra. The most extensive investigation is by Willis, who has reported upon a series of over 700 skeletons, and finds such defects in the neural arch of 4.28 per cent of individuals. It has often been stated that the neural arch of the last presacral vertebra, instead of being developed from two primary centres of ossification, may have four, one for each pedicle and superior articular process and one for each lamina, and that it is failure of the latter to fuse with the former that permits the common form of spondylolisthesis to occur. Willis suggests that although such a congenital origin is probably the only satisfactory explanation of these lesions, yet the embryological evidence upon which this statement should be based is very weak.

**What Part does Trauma take in the Development of Spondylolisthesis?**—Assuming the presence of a congenital defect, it cannot, however, be denied that trauma plays an important part in the further development of spondylolisthesis. Can this condition, on the other hand, develop as a purely traumatic lesion? The answer is at present unsatisfactory, as a study of the literature reveals that no one has presented a case in which trauma has been proved to be the causal factor to the extent that, as the result of the injury, spondylolisthesis can be shown to have developed in a spine known by previous X-ray examination to have been normal. Furthermore, the majority of cases or specimens reported upon have been advanced lesions, and no demonstration has been made of the progression in the same patient from the pre-slip stage to a well-marked spondylolisthesis. Reports have,

however, been published of cases in which the deformity has occurred in spines previously stated to have been normal clinically (Kleinberg, Turner).

It is obvious, however, that mechanical strain must be of influence, and it is probable that the greatest part is played by the ordinary influences of gravity that everyone is subjected to, though a history of violent injury to the back is presented by some patients.

Emphasis has been placed upon the increased tilt of the sacrum towards the horizontal in the production of spondylolisthesis. In fact, Armitage Whitman has described several cases of pre-spondylolisthesis in which this was a prominent factor. While not denying the existence of this and of the consequent increase in the shearing stress exerted at the lumbosacral joint, I believe that certain other details must be borne in mind. At the inception of the condition lordosis may be absent. In support of this are the following facts: The commoner types of injury complained of by those patients in whom trauma is a prominent feature are: (1) The lifting of heavy weights; (2) Falls to the ground upon the buttocks; and (3) The fall of heavy weights on to the shoulders. In none of these is lordosis likely to occur; in fact, in all of them it is an almost impossible position. In lifting a heavy weight, a man starts with his hips flexed and the lumbar spine arched backwards. Elevation of the load is largely carried out by use of the gluteus maximus

muscles with extension of the hips. Thus the sacrum is actively tilted backwards and forced against the laminae of the 5th lumbar vertebra. When an individual is forced to sit suddenly on the ground, the lumbar spine becomes flexed so that the normal curve is obliterated. If a load is suddenly forced on to the shoulders or upper back, the patient 'caves in' forwards; in other words, he collapses with the lumbar spine again held in extreme flexion. So-called traumatic spondylolisthesis usually occurs in men, in whom lordosis in extreme form is not so commonly found as in women. In the examination of the early cases in young adults where slipping has scarcely commenced, I have been impressed by the absence of lordosis. In late cases the lordosis present is, I feel, secondary to the displacement at the lumbosacral joint, and

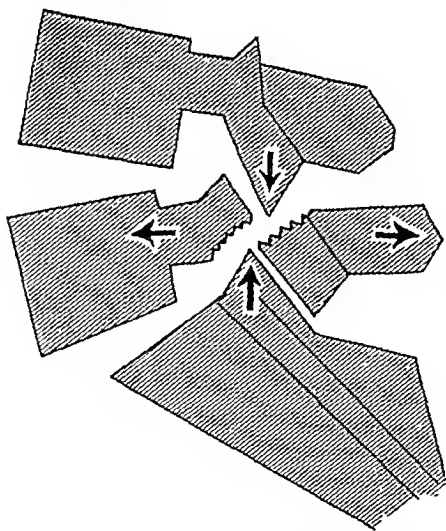


FIG. 282.—Diagrammatic lateral view of lower spine to show the influence of sacral and lumbar wedges upon the last lumbar vertebra.

is of a peculiar kind in that the sacrum, instead of being nearly horizontal, is rotated into the vertical position and carries with it the spinous process of the 5th lumbar vertebra (*see Fig. 289*).

An important factor that I think has been overlooked is the wedge-like influence provided by the upper and posterior borders of the sacrum (*Fig. 282*). The apex of the wedge is formed by the articular facets of the sacrum,

and this is driven upwards and splits the 5th lumbar vertebra into two portions at the site of congenital non-union. Thus is initiated *spondylolysis*, and 'spondylolisthesis', or slipping, follows through the continuance of the two influences of wedge action and shearing stress.

In the lumbar spine, because of the direction of its curve, weight transmission tends to be displaced posteriorly from the bodies to the laminae and articular facets, and the laminae of the 5th will thus tend to be nipped between the sacrum and

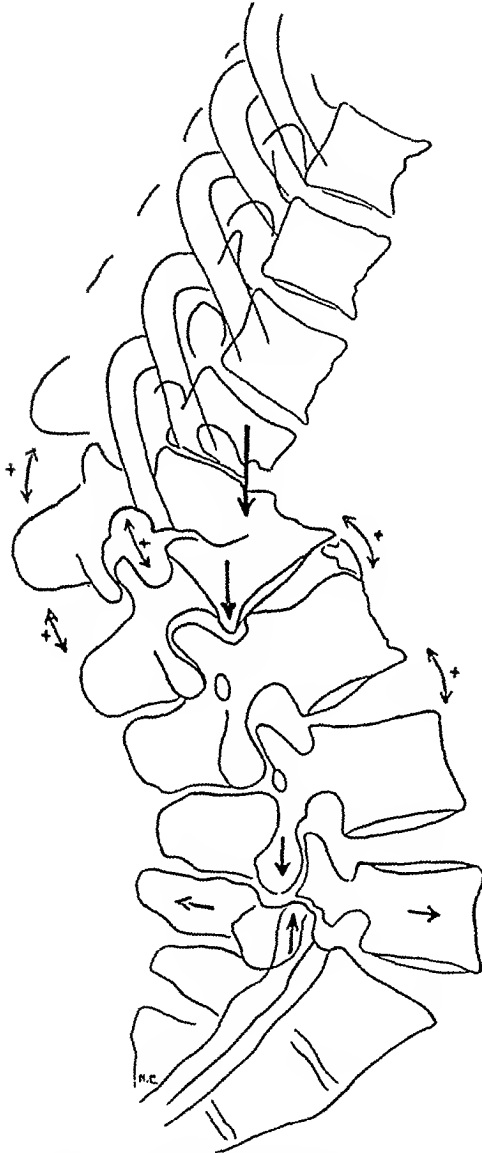


FIG. 283.—Tracing of lateral skiagram of a case of tuberculosis of the thoracic-lumbar junction. Demonstration of tensile and compression stresses and early spondylolisthesis.

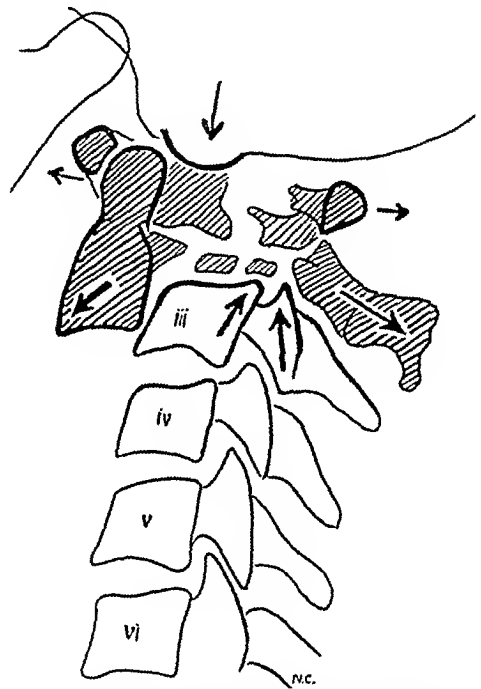


FIG. 284.—Tracing of lateral skiagram of cervical spine in a patient with fracture of the atlas and axis showing analogous splitting of vertebrae. (By courtesy of Dr. F. J. Hodges.)

an upper wedge provided by the lower articular processes of the 4th lumbar vertebra. Such conditions are difficult of demonstration in the resting spine. I am fortunate therefore in being able to present an X-ray (Fig. 283) of a lumbar spine

where these factors can be studied in a stationary form. The patient had had a long-standing tuberculous of the upper lumbar vertebrae with severe collapse anteriorly. At the lumbosacral joint a mild spondylolisthesis has commenced. Examination of the illustration, in which the chief lines of



force have been indicated by arrows, will reveal how splitting of the 5th lumbar vertebra has probably occurred.

As a demonstration of a somewhat similar wedge action elsewhere in the spine, the case illustrated in *Fig. 284* is of great interest. In this the 1st and 2nd cervical vertebrae have been split each into two halves at points analogous to the site of the lesion in spondylolisthesis. The apices of the wedges involved are here provided by the condyles of the occiput on the one hand and the anterior edges of the superior articular surfaces of the 3rd cervical vertebra on the other. The cause of this lesion was a motor-car accident.

**Spina Bifida.**—With one morphological defect, it would not be surprising to find other similar anomalies, and this in fact is true of spina bifida and the

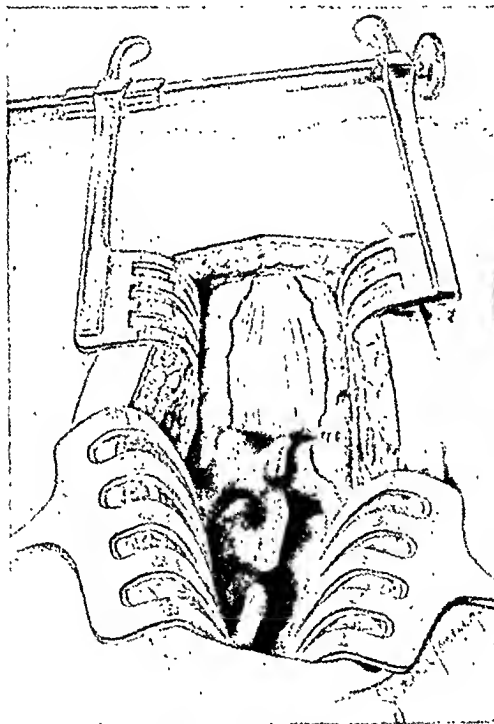


FIG. 285.—View of lumbosacral spine at operation in a case of severe spondylolisthesis, showing wide bony defect in the sacrum.

interarticular defect that is present in spondylolisthesis. While I do not believe that spina bifida bears any causal relation to spondylolisthesis, I find that those cases having wide gaps in the laminae of the 5th lumbar vertebra or sacrum do show much more severe forward displacement. In *Fig. 285* is illustrated the type of sacral defect found in two very severe cases of displacement operated upon by the writer.

**Rotato-scoliosis.**—In spondylolisthesis the amount of displacement is often not symmetrical because the laminal defect may be present on one side only.

In such cases rotato-scoliosis results (*Fig. 286*). This is of great interest, as the lumbar spine is commonly supposed to possess little power of rotation on account of the situation in relation to the sagittal plane of the articular facets. The usual reasoning regarding lumbar rotation assumes that the axis for such



FIG. 286.—Severe spondylolisthesis with rotato-scoliosis.

a movement must lie at the centre of the vertebral bodies. There are several mechanical factors that combine in the lumbar region to make it feasible for the axis of rotation to be placed posterior to the spinal canal. The centre of the circle of which the normal lumbar curve is a segment is situated posteriorly, and it is therefore easier for rotation also to occur upon a posterior

axis. If the lumbar concavity is increased, the centre of gravity is displaced posteriorly in relation to the lumbar vertebral bodies. Supposing that the axis of rotation were to be displaced to the tips of the spinous processes, the

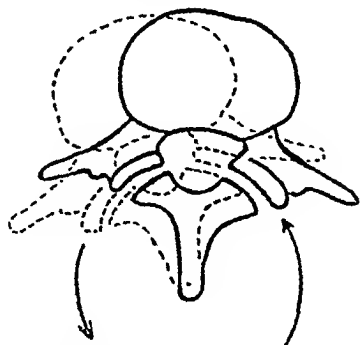


FIG. 287.—Diagrammatic outline of a lumbar vertebra demonstrating in exaggerated form possible rotation upon a posterior axis.

arrangement of the articular facets would be admirably suited to allow of a fair range of movement of this type (*Fig. 287*).

**Factors Limiting Displacement.**—That the supports of the spondylolisthetic 5th lumbar vertebra are very insecure is obvious; nevertheless in the majority of cases the displaced body comes to a final position of rest after making only a moderate forward movement. There are several factors that favour natural arrest. One of these is provided by the iliolumbar ligaments that pass from the transverse processes of the last lumbar vertebra to the iliac crests; these are assisted by the interspinous ligaments and the posterior spinal muscles. The effect of all these ligamentous structures is increased

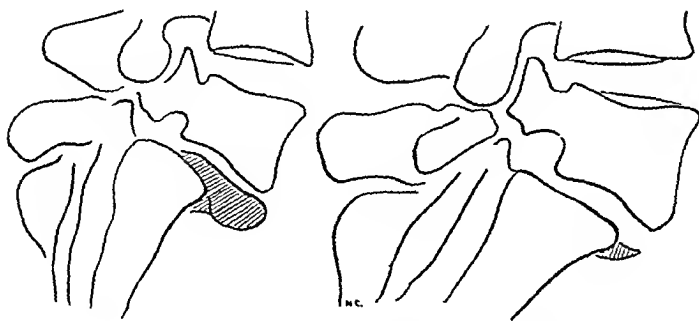


FIG. 288.—Tracings of lateral views of two cases of spondylolisthesis with buttress formation beneath the last lumbar vertebra.

by the tendency of the pelvis to assume a more vertical position by rotation on its transverse axis. Bone often proliferates from the anterior surface of the sacrum beneath the displaced portion of the 5th lumbar vertebra, forming a sufficient buttress to prevent further slip (*Fig. 288*). In many weight transmission must be partly assumed by the laminae and spinous processes, and it is possible in some for the 4th lumbar vertebra, through its inferior articular facets, to come into direct contact with the sacrum.

**Clinical Features.**—In well-advanced cases it is usually not difficult to make the diagnosis of spondylolisthesis on clinical examination alone. A

shortened trunk in which the lower ribs are depressed, sometimes almost into the pelvis, is associated with a rotation of the pelvis upon a transverse axis so that the sacrum appears more vertical (*Fig. 289*). There is a small hollow behind the lumbar spinous processes, and at the lower end of this hollow there is a bony projection which in the commonest type of spondylolisthesis is the tip of the spinous process of the 5th lumbar vertebra, and not, as often stated, the upper border of the sacrum. A peculiar waddling gait may be observed; this is due to hyperextension of the hips secondary to the pelvic rotation.

Pressure upon the branches of the cauda equina is not uncommon, and is manifested by sciatic neuralgia or neuritis. In the very early cases, low back pain is a common complaint, but nothing characteristic may be found except a small prominence of the 5th lumbar spinous process.



FIG. 289.—A patient with severe spondylolisthesis, showing characteristic rotation of the pelvis upon a transverse axis.

**X-ray Appearances.**—For early diagnosis X-ray examination is essential. In the antero-posterior view (*Fig. 290*) the well-developed cases give very characteristic appearances, as is well illustrated in Brailsford's article. The lateral view is of greater importance for early diagnosis, and the following

points at the lumbosacral junction must be observed: (1) Alinement of the anterior and posterior borders of the vertebral bodies; (2) Width of the intervertebral disc; (3) Shape of the intervertebral foramina; (4) Continuity



FIG. 290.—Antero-posterior view of a case similar to that shown in *Fig. 289*.

of the neural arches; (5) Length and relation of the spinous processes; (6) Evidence of structural bone changes; (7) Degree of lumbar curve and angle of the sacrum; (8) Evidences of spontaneous arrest.

1. In advanced cases, the forward displacement of the 5th lumbar body is easily seen. Because of the thickness of the intervertebral disc milder projections of the anterior border of the 5th lumbar body may be more difficult to distinguish. For such cases Ullmann has devised a test which is illustrated in *Fig. 291*. Regarding alinement of the posterior borders of the

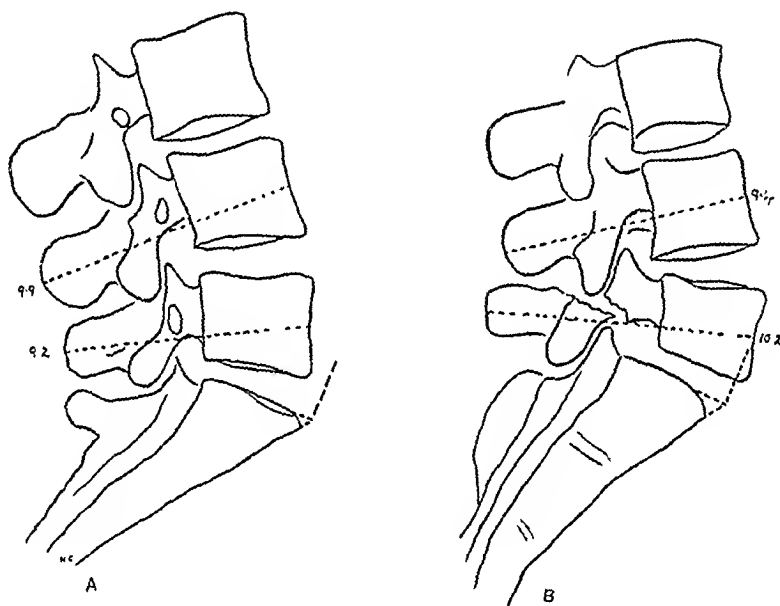


FIG. 291.—Two tests for early spondylolisthesis: (1) Comparison of antero-posterior diameters of last two lumbar vertebrae; and (2) Ullman's sign. In the latter a line is drawn at right angles to the upper border of the sacrum at its anterior edge; the 5th lumbar vertebra should lie entirely behind this line. A, Normal spine; B, Spondylolisthesis.

vertebral bodies some caution must be exercised in the interpretation of displacement, as a normal joint (because of some obliquity of the X-ray tube) may show an apparent subluxation. This is due to the shadow cast by the lateral mass of the sacrum which may project behind the posterior border of its body.

2. There is often some narrowing of the intervertebral space, which in most cases is probably secondary to the displacement; when due to the destructive lesion of tuberculosis it may be a causal factor in the origin of a spondylolisthesis.

3. The intervertebral foramina of this interval are the smallest of the lumbar series, and in spondylolisthesis are elongated irregularly in the antero-posterior direction.

4. One of the most characteristic features, which is usually seen best in early cases, is the break in the laminae; this may appear as a gap continuing the lumbosacral intervertebral clear space backwards *above* the spinous process

FIG. 292.—Untouched print of a lateral skiagram of early spondylolisthesis. The laminal defect is well shown, but the spinous processes are unfortunately lost in printing.



FIG. 293.—A skiagram of a similar case to that shown in *Fig. 292*. Certain of the bony details have been outlined in ink.

of the 5th lumbar vertebra. Occasionally this defect may be observed in the antero-posterior view (*Figs. 292-294*).

5. Normally the 5th lumbar spinous process is the shortest of the lumbar series, but in the commoner type of spondylolisthesis it projects backwards beyond the 4th spine. A useful diagnostic point is to measure the antero-posterior diameters of the 4th and 5th lumbar vertebrae. In these cases the 5th will usually be found longer than the 4th; a reversal of the normal (*see Fig. 291*).

6. As further evidence of the wedge-like influence of the sacrum there may be found, besides the separation of the lowest lumbar vertebra into two

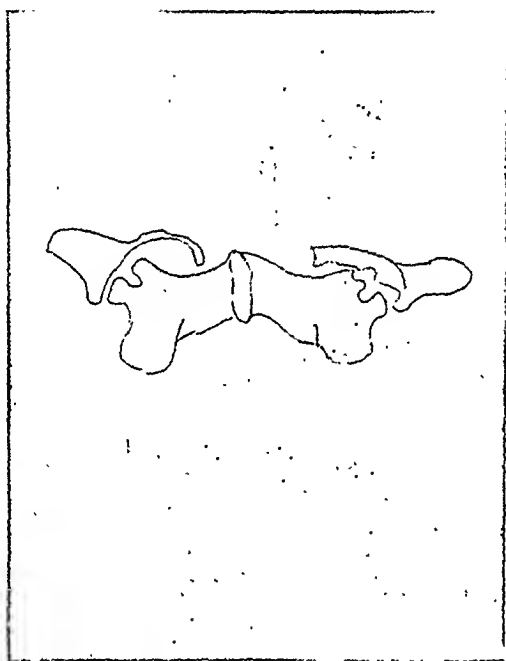


FIG. 294.—Antero-posterior view of same case as *Fig. 293*. The defective laminae have been outlined.

portions, an angulation of the two halves so that their extremities point downwards.

7. In my experience the degree of lumbar curve and the angle of the sacrum have not been excessive; as already stated, in the extreme cases the latter tends to assume the vertical.

8. As evidences of spontaneous arrest, bony buttress formation may be seen attached to the anterior surface of the sacrum under the projecting portion of the 5th lumbar vertebra, and the lower articular process of the 4th may rest on the sacrum (*see Fig. 288*).

**Treatment.**—That many patients have spondylolisthesis without symptoms cannot be denied. In the early cases the temptation is great to try to prevent the further development of the deformity by bone-grafting operations. The

early treatment should, however, be that of any postural backache: the removal of conditions of labour likely to accentuate the lesion, a period of rest, and the provision of a brace where necessary. Operation should be reserved for cases not responding to such treatment or which are giving evidence of further slipping. Bone-grafts undoubtedly assist many patients, but it must be remembered that the result does not, from the mechanical point of view, possess great efficiency. Tremendous forces are brought to bear upon the graft, which is badly placed for the prevention of further slipping. In cases with sacral spina bifida it is difficult to obtain fixation for the graft at the lower end (*see Fig. 285*). The ideal operation would be either an anterior bone-graft so placed as to fix the body of the 5th lumbar vertebra to the sacrum and forming a buttress, or some form of antero-posterior fixation of the two halves of the divided vertebra (*Fig. 295*). The technical difficulties of such procedures, however, preclude their trial.

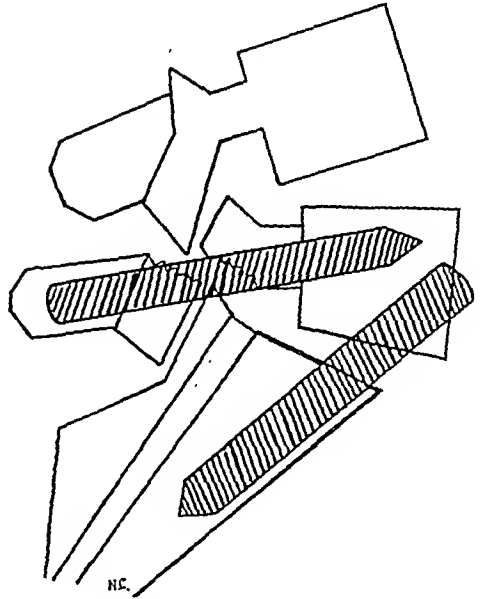


FIG. 295.—Theoretical alternatives to the posterior bone-graft operation for spondylolisthesis.

### SUMMARY.

Spondylolisthesis, equally common in men and women, is found to be of two types. The more usual results from a separation, through a probable congenital defect, of the 5th lumbar vertebra into two portions. Besides the forward displacement of the body of this vertebra, its spine and laminae are displaced backwards with the sacrum.

The place of trauma in producing the subluxation and the influence of other factors besides lordosis are discussed. The author believes that the sacrum acts as a wedge, the apex of which (the upper and posterior edge) is driven upwards and causes a diastasis of the last lumbar vertebra, and that the posterior portions of the lumbar vertebrae take a more active part in the production of spondylolisthesis than is usually recognized.

Factors favouring natural arrest of the displacement are described; the surest of these is the formation of a bony buttress from the front of the sacrum.

Good lateral X-ray studies are frequently the only means of detecting the very early displacements, which will be found to be more common than is usually suspected. Roentgenological tests for early cases are illustrated.

Mechanically ideal, though technically unsuitable, operative procedures



are mentioned and the usual treatment is outlined; this in most cases should first be conservative.

I wish to acknowledge my indebtedness to the late Dr. P. M. Hickey, Professor of Roentgenology at the University of Michigan, who inspired me to carry out this study and who generously placed the resources of his department at my disposal.

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For more complete bibliographies, the reader is referred to the articles by Fitch, Brailsford, and Faldini.

**POST-OPERATIVE MASSIVE COLLAPSE OF THE LUNG.\*****A CLINICAL AND EXPERIMENTAL STUDY.**

BY DAVID BAND AND I. SIMSON HALL, EDINBURGH.

**INTRODUCTION.**

MODERN technique in operative surgery has brought about a great drop in the incidence of complications to surgical cases in the post-operative period. Improved anaesthesia, general or local, pre-operative preparation, the anticipation and treatment of shock, asepsis, gentleness in handling, careful dissection, and intelligent and co-operative after-treatment have all followed increased knowledge of surgical pathology, living pathology, and biochemistry.

With the diminution of the incidence of sepsis, shock, cardiac failure, etc., attention has become drawn to the increasing number of cases in which pulmonary complications are present. In this group are many classes of lesion, but their increasing importance is seen from these figures taken from Cutler, who found the reported incidence of pulmonary complications after operation to be :—

1898	..	..	0.38 per cent
1916	..	..	1.86 per cent
1921	..	..	3.5 per cent and a mortality of 0.5 per cent

That author, from the records of 1562 cases, estimated the case-mortality of a post-operative pulmonary complication at 20 per cent. Of these chest conditions, 76.4 per cent are noted as commencing within forty-eight hours of operation, and they are described under the diagnoses of pleurisy, bronchitis, bronchopneumonia, lobar pneumonia.

In other analyses of pulmonary post-operative complications similar figures are obtained, and the lesions are described under similar headings. One author, Beekman<sup>2</sup> (1910), has given a double analysis of circulatory and lung complications, and it is interesting to note that his figures for phlebitis and pleurisy are closely approximate, viz., 0.38 per cent and 0.33 per cent, almost 60 per cent of the former having the further and more deadly complication of pulmonary embolism. These last figures are of special interest when it is remembered that Cutler, Cutler and Hunt,<sup>11</sup> and Schluter and Weidlem, in a series of papers, have developed the embolic theory in relation to these pulmonary complications. Bronchitis and bronchopneumonia are alleged to be due to showers of minute emboli released from the lymphatic

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field of the operation area, carried in the lymphatic circulation to the blood-stream, thence to the right heart and lung. A mild pneumonitis is set up, and secondary factors of sepsis, blood-borne or inhaled, determine the severity of the subsequent bronchopneumonia. In animal experiments they were able to control the effect of an embolus artificially introduced via the jugular vein. Different degrees of infecting virus in the artificial embolus produced a mild or virulent pneumonitis; the milder strains, a pulmonary abscess. They blame local chilling, sepsis, asphyxia, acidosis, and the primary disease as the main factors in etiology, while rough handling and irritative anaesthetics also play an important rôle.

The use of the X rays in chest diagnosis, and a closer investigation of the post-operative chest from a clinical standpoint, probably account for the increasing number of collapse cases now reported; so that we have Scott<sup>32</sup> (1925) describing no fewer than forty cases of massive atelectasis. The tabulated facts are:—

*Seasonal Influence*.—nil. *Age*.—12 to 49 years; 40 per cent third decade. *Time*.—80 per cent within 48 hours of operation. *Duration*.—2 to 17 days. *Result*.—in this series all recovered. *Site*.—75 per cent right lung, usually lower lobe.

*Description*.—Pain, dyspnoea, tachycardia, cyanosis, cough. Temperature 100°–104·8°. Pulse 108–160. Respiration 30. Thoracic asymmetry—unilateral dullness and displacement of heart to the affected side. Breath-sounds: (1) Diminished, very faint bronchial breathing; (2) Coarse râles.

*X-ray Appearance*.—Marked density of affected lung, deviation of heart to affected side, elevation and relative immobility of the diaphragm.

Following the appearance of râles the signs become those of a bronchopneumonia with a thick tenacious sputum.

The types may be classified as: (1) Fulminating; (2) Moderately severe; (3) Latent. They terminate by: (1) Lysis; (2) Complications; (3) Crisis.

In a recently published article on the subject Sheret<sup>34</sup> reports the occurrence of pulmonary collapse in 18·1 per cent of 149 male patients and 13·7 per cent of 112 female patients in their post-operative course.

The condition of massive atelectasis of the lung was recognized by Legendre, Bailey, and others as far back as the middle of the last century. Since that time it received but spasmodic attention till, in 1890, Pasteur<sup>29</sup> described the condition and by his later work established it on a sound basis as a clinical entity.

From this time many complete accounts have been given, notably by Scott,<sup>32</sup> Churchill,<sup>8</sup> Leopold,<sup>25</sup> and others, so that any further complete summary would serve no useful purpose. Perusal of the literature, however, convinces us that, excellent as many of these articles are, the evidence upon which rest the many and various theories advanced is extremely slender when it is judged from experimental or clinical standpoints. The present series of experiments was undertaken to attempt to supplement that evidence and provide a sound basis of experimental proof for one theory of the origin and mechanism of the condition, when met with in its post-operative form, and possibly at the same time to provide some link with other types which at first sight appear unrelated to that just mentioned.

There is no doubt that we owe our knowledge of the ultimate mechanism of collapse to the classical experiments of Lichtheim,<sup>26</sup> whose work, quoted

and described by practically every writer on this subject, proved beyond doubt that the collapse of the alveoli was due to the absorption of alveolar air into the blood-stream. Hence all investigation is directed towards the discovery of conditions or mechanisms which will tend to this result.

The action of drugs on the bronchi has frequently been investigated with a view to ascertaining whether these can play any part in producing an obstructive or tonic condition in the bronchial muscle. Dixon and Brodie<sup>12</sup> in 1903 gave a very complete account of the effects of drugs on bronchial muscle. They point out that chloroform, ether, and atropine tend to dilate the bronchioles, while suprarenal extract produces slight contraction. They further state that no tonic effect was ever observed. Bullowa and Gottlieb<sup>6</sup> mention similar observations carried out with the help of the fluorescent screen and radio-opaque substances.

The diaphragm has been investigated by various experimenters, in a number of cases with the object of disproving Pasteur's original statement that massive collapse was due to the paralysis of the diaphragm. Elliott and Dingley<sup>13</sup> early established the fact that no collapse of the lung follows division of the phrenic nerve or hemisection of the cord at the second or third cervical segments. Hemisection produces paralysis of the intercostals as well as of the diaphragm, in effect depriving the lung on the one side of the chief part of its movement. Schroeder and Green<sup>31</sup> give a very complete account of the effect of phrenicectomy, and add the interesting conclusion that, although there is an innervation of the margin of the diaphragm from the intercostal nerves, this supply is in no way sufficient to carry on the work of the diaphragm after section of the phrenic nerve. Coryllos and Birnbaum<sup>9</sup> also have failed to produce atelectasis after division of the phrenic nerve. More recently Lemon<sup>24</sup> has shown that, although diaphragmatic excursion is affected after phrenic neurectomy, there is no influence on vital capacity.

The influence of the extrinsic nerves of the lung has been dealt with by Fontaine and Herrmann,<sup>16</sup> who succeeded in denervating the lungs of dogs almost completely. Their results are of importance from the fact that they tend to show that there followed no change in respiratory movement, and that severing of the nervous connections did not prevent collapse.

The most interesting experiments, bearing on atelectasis as a post-operative complication, are those of Tucker, Lee, and their co-workers.<sup>22, 23, 35, 36</sup> By simulating the conditions found by actual observation through the bronchoscope in patients suffering from atelectasis, they have reproduced the condition and have been able to study its mechanism and treatment. The mechanism of onset and the appearances of the lung during the production of the atelectasis, as well as the influence of various gases, have been recently studied in an exhaustive manner by Coryllos and Birnbaum. By an ingenious balloon they have been able to occlude the lung at will, and so study the process of atelectasis in detail. The experiments of Lee and Tucker, however, have gone a step farther by attempting to produce, not so much the condition of atelectasis by a known process, but the conditions predisposing to it, and observing the reaction of the lung to these conditions and to the methods of treatment.

## REPORT OF FOUR CLINICAL CASES OF POST-OPERATIVE PULMONARY ATELECTASIS.

We are indebted to Professor D. P. D. Wilkie for permission to record the following cases.

*Case 1.*—J. B., male, age 33 years. This patient was admitted to the Royal Infirmary, Edinburgh, with a history of indigestion over a period of ten years. A diagnosis of duodenal ulcer was made following clinical and radiological investigation. He had been gassed during the war, otherwise the general health had always been good, and no morbid sign was present in the other systems.

**OPERATION** (Anæsthetic: chloroform and ether).—Transverse epigastric incision. A chronic peptic ulcer was present on the postero-superior aspect of the first part of the duodenum. There was a mucocoele of the appendix. A gastro-duodenostomy was performed, and the appendix was removed. Next day the patient appeared distressed, the respiratory rate had increased to thirty per minute, and the pulse and temperature were elevated. Some cyanosis was present. The heart was displaced to the right, and dullness and diminished breath-sounds were present at the right base. On X-ray examination marked density was found over the lower half of the right lung, and the heart and mediastinal shadows were displaced to the right. Carbon-dioxide gas inhalations were administered, and brought about rapid improvement in the patient's condition. Respirations became deeper, the cyanosis disappeared, and the expectoration became more abundant. In two days the pulse and respirations had returned to normal, though dullness, coarse râles, and bronchial breathing were present in the chest. Complete return to normal, as seen in a radiogram, did not occur till the eleventh day after the operation. The patient was discharged from hospital on the twenty-first day after the operation.

*Case 2.*—Mrs. E. D., age 55 years. Patient had had digestive symptoms for thirty years. She was admitted to the Royal Infirmary, Edinburgh, four hours after perforation of a gastric ulcer.

**OPERATION** (Anæsthetic: chloroform and ether).—Right paramesial incision. A perforated gastric ulcer was present on the lesser curvature of the stomach. Free fluid was found in the peritoneal cavity. The perforation was closed and a glass suprapubic drain was inserted to the pouch of Douglas. Recovery was uneventful till the fifth day, when respiratory distress commenced, accompanied by cyanosis and elevation of temperature and pulse. The physical signs were: dullness at the left base, diminished breath-sounds, and movement of the heart to the left side. The diagnosis of massive collapse was confirmed by the radiogram, which demonstrated marked density of the left lower lobe of the lung and movement of the heart shadow to the affected side. With carbon-dioxide therapy relief was brought about in forty-eight hours, though physical signs in the lung persisted for nearly three weeks. The heart shadow remained slightly displaced for sixteen days after the onset of the condition.

*Case 3.*—F. R., female, age 13 years. Patient had two mild attacks of appendicitis during the preceding year. On the morning of admission to the Royal Infirmary, Edinburgh, she was seized with severe abdominal pain, which passed to the right iliac fossa. Superficial and deep tenderness were present over McBurney's point.

**OPERATION** (Anæsthetic: chloroform and ether).—Gridiron incision. The appendix was acutely inflamed and pelvic in position. The appendix was removed with drainage. Recovery was uneventful till the fifth day after operation, when the patient became slightly cyanosed and developed a cough. The left lung base was dull to percussion and breath-sounds were faintly bronchial in character. Radiological examination demonstrated massive collapse of the left side (*Figs. 296, 297*). Later the same day some sticky mucus was coughed up, and thereafter the breath-sounds became vesicular and crepitations were heard. No elevation of temperature occurred. Two days later the chest was clear, both on clinical and radiological examinations.

FIG. 296.—*Case 3.* Forty-eight hours after the onset of symptoms. Marked density throughout the greater part of the left lung. The heart is displaced to the left. The left half of the diaphragm is elevated.



FIG. 297.—*Case 3.* Nine days after the onset of symptoms. The lung densities are equal and normal. The heart shadow has returned to its normal position. The diaphragmatic levels are equal.



*Case 4.*—G. S., male, age 22 years. Patient was a healthy young man who had suffered from intermittent attacks of abdominal pain associated with nausea and vomiting. Slight tenderness on deep palpation over McBurney's point persisted, and a diagnosis of chronic appendicitis was made.

**OPERATION** (Anæsthesia: chloroform and ether).—Battle's incision. The appendix was kinked and bound down by numerous adhesions and its wall was thickened. The appendix was removed. The following day the patient showed very marked signs of respiratory distress. Cyanosis was present, and there was elevation of pulse and temperature. Dullness was present over the greater part of the right lung, and breath-sounds were faintly bronchial in character. The cardiac dullness had moved to the right of the sternum. On radiological examination



FIG. 298.—*Case 4.* Forty-eight hours after the onset of symptoms. Marked density of lower lobes of the right lung. Heart displaced to the right.

marked density was present throughout the lower two-thirds of the right lung, and the cardiac shadow was seen drawn over to the right (*Fig. 298*). The patient remained very distressed, cough was persistent and non-productive. Many coarse râles could be heard throughout the affected lung. On the third day after the development of the collapse the condition of the patient was very grave. Expectoration was very slight and coughing very ineffectual in getting rid of the bronchial secretion. Bronchoscopic aspiration of the retained mucopurulent secretion was carried out, after spraying the upper respiratory passages with cocaine. On passing the bronchoscope through the cords, the deviation of the trachea to the right became apparent by a slight bulging of the left tracheal wall. When the bronchoscope was passed down to the level of the carina this was found to the right of the mid-line of the visual field. The mouth of the right bronchus contained a considerable quantity of whitish secretion. About 10 c.c. of thick viscid secretion was removed from the right lung. Movement in the right lung could not be observed accurately owing to the amount of secretion lying in the bronchi, but movement was obviously diminished. There was immediate relief to the respiratory distress, and the pulse and temperature fell. The breath-sounds became bronchial in character, with finer accompanying râles. Recovery was complete in ten days following the bronchoscopic aspiration, and the chest was normal both in clinical and radiological examinations.

On bacteriological examination the sticky mucopurulent secretion aspirated was found to contain a heavy infection of pneumococci. The glutinous character of the material was particularly noticeable. Part of the secretion was laid aside for experimental injection; the remainder was tested against various dilutions of gum acacia, in order to get a measure of its viscosity. The viscosity of the secretion was estimated at 75 per cent by comparison with similar strengths of gum acacia.

After this experience it seemed appropriate to follow the example of Tucker and Lee,<sup>22, 23, 35, 36</sup> and try to produce what seemed to us the predisposing conditions under which we might expect the appearance of collapse. To do this we have used similar methods, utilizing gum solutions to simulate secretion in the bronchi.

### THEORIES OF CAUSATION.

Theories of the origin of atelectasis fall roughly into four groups: (1) Those which postulate an active process in the lung, probably of reflex nervous origin; (2) Those in which posture is considered the most important point; (3) Those in which the essential is an absorption of air below an obstruction such as would be provided by a plug of mucus; (4) Diaphragmatic paralysis. Several writers put forward a combination of these possible causes and suggest various underlying states to account for them.

Pasteur<sup>29</sup> believed that atelectasis was due to an active contraction of the lung following the paralysis of the diaphragm, the contraction being of the nature of an imbalance between the contractile and expansile powers of the lung owing to the impairment of the latter by diaphragmatic failure. Elliott and Dingley,<sup>13</sup> however, very shortly afterwards pointed out that the arrangement of the lung musculature does not permit of this supposed action.

One of the most popular theories is that spasm or some vasomotor disturbance is at work. Scott,<sup>32</sup> Santec,<sup>30</sup> Leopold,<sup>25</sup> Seringer,<sup>33</sup> Bergamini and Shepherd<sup>3</sup> all incline to the view that bronchial spasm is the underlying condition. Seringer draws attention to the fact that the smooth muscle fibres in the bronchial walls are scanty, and he believes that they are insufficient to cause a complete collapse, and, that being so, he assumes that the presence of mucus is required to complete the mechanism. Santec, Bergamini, and Shepherd produce the most convincing arguments for some such mechanism in their quotations of four cases, with complete pathological reports of death at the time of operation, the cause being massive atelectasis.

Apart from this clinical evidence, and in passing, we may remark that we are not convinced, as the authors seem to be, that the obstructive theory is inapplicable; there is singularly little evidence that reflex irritation or spasm can produce anything resembling atelectasis—in fact, the experimental evidence goes far to negative the possibility. Opening the abdomen and traumatizing the viscera has never been observed to cause any spasm of the bronchi, and violent external stimuli seem to act by fixation of the muscles of respiration, and in this way interfere with the normal mechanism of the lung.

The work of Fontaine and Herrmann<sup>16</sup> on denervated lungs points to the fact that if there is any question of nervous stimulus, the cause is just as likely to be found in the lung itself as in the operation field. Again, we may refer to the fact that so far we have no record of cases of collapse of the lung in those conditions which we are accustomed to refer to as of nervous origin, namely, asthma and angioneurotic oedema. Indeed, these affections have been proved to cause the reverse condition, namely, emphysema, by virtue



of the intermittent spasm. As far as we are aware, there is no record of the production of tonic spasm of the bronchi either by clinical or experimental observation. We cannot help concluding, therefore, that there is no direct evidence that any condition of spasm or other vasomotor disturbance is produced in the lung which is concerned with the causation of massive atelectasis.

One of the most difficult classes of case to reconcile with the more favoured theories of causation is the traumatic type—for example, that following gunshot wounds of the chest. Sir John Rose Bradford<sup>4</sup> has given a very complete account of the atelectasis which accompanies a considerable percentage of all penetrating wounds of the chest. This writer mentions that urgent symptoms have always been absent in contralateral collapse, but also observes that no case of complete contralateral collapse has been seen. This seems to indicate a process of gradual onset, or one in which the symptoms are masked by the greater trauma of the wound, and it also lends support to Crymble's<sup>10</sup> observation that the wounded man tends to lie on the unwounded side, thus introducing the very definite factor of limitation of lung movement. Crymble further points out that the recovery of the collapse is independent of the processes in the opposite lung, meaning that, although the other lung becomes more involved by pathological processes, the recovery of the collapsed lung is not unduly retarded. Here again we have the condition differing apparently in pathology from the trauma it accompanies, and pointing to some factor which is coincident with the wound. Lilienthal makes the suggestion that the pathology of gunshot collapse differs from that of post-operative collapse, but it seems unnecessary to complicate the matter in this way, as the pathology itself has been determined by Liehtheim,<sup>26</sup> Coryllos and Birnbaum,<sup>9</sup> and others, the mechanism by which it is produced being the problem which requires solution.

Pursuing the subject of posture as a possible factor in the production of atelectasis, we find in Briscoe<sup>5</sup> a strong supporter of this theory, and he has worked out several different types of patient likely to develop atelectasis from the deflation of the lower lobes consequent on the posture adopted, the determining factor being crural disability from overlying pleurisy. The theory is interesting in that the ultimate mechanism is purely diminution of respiratory force. While it is feasible for certain cases, the chief objection seems to us to lie in its inapplicability to so many other types of case. The assumption of the presence of pleural inflammation in every individual suffering from this complication appears to us to be unwarranted.

In conclusion, one would like to refer to those cases which have been recorded as occurring after operation under spinal anaesthesia. These have been pointed out as disproving the fact that massive collapse is primarily due to lung interference. We think, however, that these cases are strongly in favour of the occurrence of some such mechanism as we describe, as will be seen if the technique of this particular form of anaesthesia is considered. Anaesthesia frequently reaches above the costal margin, and the intercostal muscles are out of action when the abdomen is opened. The consequence is that the diaphragm has to carry the whole burden of respiration. If the operation is upper-abdominal, there is considerable interference with the diaphragm by handling. If lower-abdominal, the probability is that some

degree of Trendelenburg position is used, thus causing further embarrassment of the diaphragm by the tendency of the viscera to move to the upper part of the abdominal cavity.

Thus we have the essential mechanical factor of serious embarrassment of respiratory mechanism.

## EXPERIMENTAL INVESTIGATION.

Using dogs, an extensive series of operative procedures was carried out, individually and in combination, under narcosis. The series of experiments has been divided into nine groups, each of which introduced a variation in the operative procedure or in the technique employed. The effect on the respiratory tract was studied in each group, using clinical, bronchoscopic, and radiographic methods. In certain experiments the animal was killed, and the respiratory organs were examined at autopsy. The groups were made up as follows:—

### Group

- I.—Laparotomy.
- II.—Bronchoscopy and bacteriological examination of normal dog's bronchus.
- III.—Bronchoscopy and the introduction of gum acacia of varying degrees of viscosity to the right bronchus.
- IV.—Bronchoscopic introduction of a solid foreign body to the lumen of the right bronchus.
- V.—Laparotomy combined with bronchoscopic introduction of gum acacia to the lumen of the right bronchus.
- VI.—Bronchoscopic introduction of gum acacia of low viscosity, followed by strapping of the chest.
- VII.—Introduction of gum acacia of viscosity similar to specimen of bronchial content obtained from a patient suffering from massive collapse, followed by the application of adhesive strapping to the lower ribs.
- VIII.—Exposure of the right phrenic nerve in the neck, study of the effect of electrical stimulation and avulsion of the nerve.
- IX.—Bronchoscopic introduction of gum acacia in a previously phrenicectomized animal.

Narcosis was produced by the hypodermic injection of amytal and morphine two hours prior to the experiment. The dosage was calculated from the body weight of the animal. The anaesthesia obtained gave complete satisfaction for all operative procedures, and the cough reflex was abolished. The return to consciousness was complete in three to three and a half hours from the time of the injection, and no undesirable symptoms presented. By avoiding an inhalation anaesthetic, the intrabronchial secretion was under the control of the operators and bronchoscopic examinations were made with greater freedom. Massive collapse of the lung developed in Groups VII and IX in every case.

## EXAMPLES FROM TYPICAL ANIMAL EXPERIMENTS.

### Group I.—Laparotomy.

Dog, male, weight 10.45 kilos.

Preliminary X-ray examination of the chest revealed no abnormality. The lungs were of equal and normal density. Both halves of the diaphragm moved symmetrically.

Anæsthesia : open ether.

Abdomen opened by a paramesial incision through the right rectus muscle. The stomach, liver, and small intestine were handled and slightly traumatized. The wound was closed in layers with linen sutures.

In 24 hours : X-ray examination repeated. Lung fields of normal density, diaphragmatic movement normal and equal. Heart shadow undisplaced. No lobular collapse of the lung present. Recovery uneventful.

*Group II.—Bronchoscopy.*

Dog, male, weight 10.45 kilos.

Anæsthesia : amytal 6 c.c. ; morphine 1 gr. hypodermically, given two hours prior to experiment.

Bronchoscope passed. Bronchi inspected and found to be normal. Swabs taken from bronchial mucosa and from teeth for bacteriological examination. Movements of bronchial tubes during inspiration and expiration studied.

Lipiodol was injected through the bronchoscope. The animal was X-rayed and a normal photograph of the bronchial tree obtained.

BACTERIOLOGICAL REPORT.—Culture from swab from normal dog's bronchus yielded growths of a few streptococci, a few pneumococci. Culture of swab from healthy dog's teeth yielded growths of a few short strepto- and diplococci.

*Group III.—Bronchoscopy and the introduction of gum acacia of varying degrees of viscosity to the right bronchus.*

a. Dog, male, weight 11 kilos.

Anæsthesia : amytal 5.5 c.c. ; morphine 1 gr.

Bronchoscope passed, and 5 c.c. of 40 per cent gum acacia injected into the lumen of the right bronchus.

The animal showed no signs of distress and appeared quite normal twenty-four hours later. X-ray examination the following day revealed a few scattered areas of lobular collapse at the base of the right lung. There was no displacement of the heart.

b. As in previous experiment.

Ten c.c. of 50 per cent gum acacia injected into the lumen of the right bronchus.

Result similar to that in previous experiment. A few areas of lobular collapse were noted.

*Group IV.—Bronchoscopic introduction of a solid foreign body to the lumen of the right bronchus.*

Dog, male, weight 11.4 kilos.

Anæsthesia : amytal 5.5 c.c. ; morphine 1 gr.

A solid portion of rubber reinforced by a U-spring was accurately fitted into the lumen of the right bronchus, to remain there securely after release of the spring. X-ray examination carried out shortly afterwards revealed the foreign-body plug to be in position (*Fig. 299*). On recovery from the anæsthetic the animal was active, and coughed considerably. Twenty-four hours later



*Fig. 299.*—*Group IV.* The foreign body is in position in the right bronchus. Heart and lungs normal.

## POST-OPERATIVE COLLAPSE OF LUNG 397

X-ray examination revealed the foreign body in the animal's stomach. The lung was normal and the animal well.

*Group V.*—Laparotomy combined with bronchoscopic introduction of gum acacia to the lumen of the right bronchus.

*a.* Dog, male, weight 12 kilos.

Anæsthesia : amytal 6 c.c. ; morphine 1 gr.

Bronchoscopic injection of 10 c.c. of 50 per cent gum acacia to the lumen of the right bronchus.

Laparotomy carried out immediately through a right rectus incision. The abdominal viscera were handled, and the wound closed in layers by linen sutures. A slight degree of dyspnœa was noted after operation. Twenty-four hours later the animal appeared normal. On X-ray examination scattered areas of lobular collapse were present in the right lung.

*b.* Dog, male, weight 11.3 kilos.

Anæsthesia : amytal 5.5 c.c. ; morphine 1 gr.

Laparotomy carried out as in previous experiment. Bronchoscopic introduction of 15 c.c. of 75 per cent gum acacia to the lumen of the right bronchus. The animal became dyspnœic and remained with signs of respiratory distress for three hours after the experiment. Twenty-four hours later the animal appeared normal. On X-ray examination there was a considerable number of scattered areas of lobular collapse in the right lung. There was no deviation of the heart or the mediastinum.

*Group VI.*—Bronchoscopic introduction of gum acacia of low viscosity, followed by strapping of the chest.

Dog, male, weight 11 kilos.

Anæsthesia : amytal 6 c.c. ; morphine 1 gr.

Six c.c. of 40 per cent gum acacia were introduced to the lumen of the right bronchus. A band of adhesive strapping was applied round the lower ribs.

Twenty-four hours later X-ray examination revealed considerable lobular collapse in the right lung, but no deviation of the heart.

*Experiment : BRONCHOSCOPIC INTRODUCTION OF BRONCHIAL CONTENTS FROM A PATIENT SUFFERING FROM POST-OPERATIVE MASSIVE COLLAPSE OF THE LUNG.*

Dog, female, weight 7.4 kilos.

Anæsthesia : amytal 4.5 c.c. ; morphine 1 gr.

Following introduction of the bronchoscope, 3 c.c. of the bronchial content, which had been preserved with aseptic precautions, were injected into the lumen of the right bronchus. In ten minutes it was noted that there was a slight increase in the respiration rate in the animal, but thirty minutes later a return had been made to normal. Fifteen hours later animal found dead.

*Post-mortem Report.*—Generalized bronchopneumonia was present. A pure growth of pneumococci was obtained from bacteriological examination of uncontaminated lung tissue.

*Group VII.*—Introduction of gum acacia of viscosity similar to specimen of bronchial content obtained from patient suffering from massive collapse, followed by the application of adhesive strapping to the lower ribs.

*a.* Dog, male, weight 12 kilos.

Anæsthesia : amytal 6 c.c. ; morphine 1 gr.

Bronchoscope introduced and the injection of 75 per cent gum acacia continued until the lumen of the right bronchus was filled, 30 c.c. in all. A strip of adhesive strapping was then applied to the lower ribs.

In 3 hours : slight increase in density of right lung, associated with some displacement of the heart to the right.

In 24 hours : complete and marked density throughout right lung. Heart and mediastinum displaced to the right. The right half of the diaphragm is elevated. The animal is restless and inclined to be savage. (*Fig. 300.*)



FIG. 300.—*Group VII (Dog a)*. Twenty-four hours. Complete density throughout right lung. Heart and mediastinum displaced to the right. Right half of the diaphragm elevated. Intra-bronchial gum acacia and strapping of the chest.



FIG. 301.—*Group VII (Dog a)*. Forty-eight hours. Marked density of right lung. Heart displaced to right.



FIG. 302.—*Group VII (Dog a)*. Ten days. The heart is normal in position. The lung is clear except for one or two areas of lobular collapse.

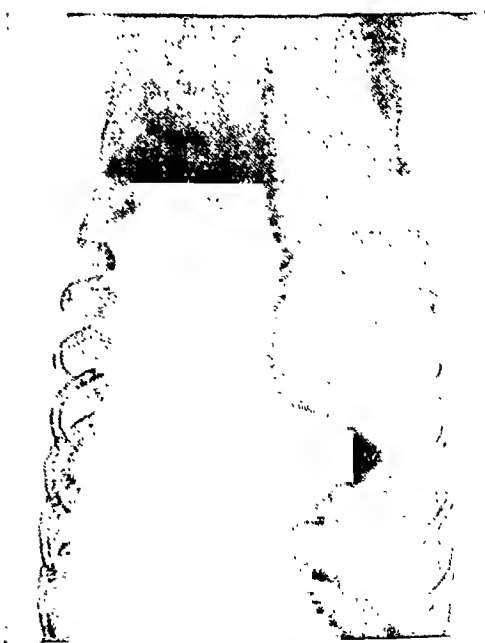


FIG. 303.—*Group VII (Dog b)*. Twenty-four hours. Complete density through right lung. Heart and mediastinum displaced to right. Right half of diaphragm elevated. Intra-bronchial gum acacia, and strapping of the chest.

In 48 hours: marked density throughout the right lung. Heart displaced. (Fig. 301.)

In 4 days: upper and middle lobes still dense. Lower lobe clearing. Heart still displaced and diaphragm elevated.

In 8 days: heart shadow has almost returned to normal position. Lower part of lung clearing rapidly. There are still scattered areas of increased density. Diaphragm functioning.

In 10 days: heart normal in position: still slight lobular collapse in lung. (Fig. 302.)

b. Dog, male, weight 10.5 kilos.

Anæsthesia: amytal 5 c.c.; morphine 1 gr.

As in previous experiment. Introduction of bronchoscope and injection of 20 c.c. 75 per cent gum acacia into right bronchus. A strip of adhesive strapping was applied to the lower ribs. Early dyspnoea. X-ray examination.



FIG. 304.—Group VII (Dog b). Six days. The density over the right lung is clearing irregularly. The right half of the diaphragm is still elevated. The heart has not yet returned to its normal position.



FIG. 305.—Group VII (Dog c). Complete density throughout right lung. Displacement of the heart to the right. Elevation of right half of the diaphragm. The animal was killed at this stage. Intrabronchial gum acacia and strapping of the chest.

In 24 hours: displacement of heart to the right; marked density over whole of right lung; right half of diaphragm elevated. (Fig. 303.)

In 3 days: heart shadow has returned slightly in normal direction. Marked and persistent density over whole of right lung.

In 6 days: density over right lung is clearing irregularly. The diaphragm is still elevated. The heart has not yet returned to its normal position. (Fig. 304.)

In 11 days: heart and diaphragm normal in position and appearance. Lung clear except for one or two slight areas of lobular collapse.

c. Dog, female, weight 9.4 kilos.

Anæsthesia: amytal 5 c.c.; morphine 1 gr.

Bronchoscopic introduction of 20 c.c. of 75 per cent gum acacia to right bronchus. Application of strapping to lower ribs. X-ray examination.

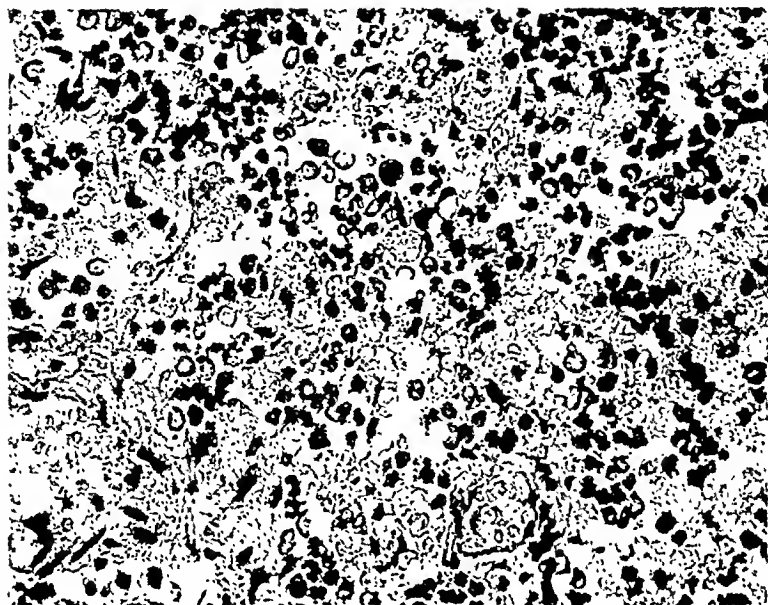


FIG. 306.—*Group VII* (Dog c). Section of right lung twenty-four hours after induction of collapse. The alveoli are collapsed, the lining cells are cedematous, and a hæmorrhagic exudate has occurred. The vessels are greatly dilated.



FIG. 307.—*Group VII* (Dog c). Section of normal left lung.

# POST-OPERATIVE COLLAPSE OF LUNG 401

In 24 hours: complete density over right lung, displacement of heart to the right, elevation of diaphragm. (Fig. 305.)

The animal was killed with chloroform and autopsy at once carried out. The right lung was found to be smaller than normal, darkly congested, without normal crepitus, and heavier than water. The left lung was normal in all respects.

Sections were cut for histological study. These demonstrated alveolar collapse throughout the affected lung; there was in addition considerable congestion. Dilatation of the vessels was present and a hæmorrhagic exudate had occurred throughout. (Figs. 306, 307.)

In this group massive collapse of the lung was experimentally produced in every case.

*Group VIII.*—Right phrenic nerve exposed in the neck and divided, electrical stimulation, and later avulsion of the distal end carried out.

Dog, female, weight 9 kilos.

Anæsthesia: amytal 5 c.c.; morphine 1 gr.; open ether.



FIG. 308.—*Group VIII.* Effect of electrical stimulation of right phrenic nerve. The right half of the diaphragm is in contraction.



FIG. 309.—*Group VIII.* Effect of avulsion of the right phrenic nerve. The right half of the diaphragm is elevated and paralysed.

Oblique incision made in the neck: cervical nerves and phrenic nerve exposed and isolated, right phrenic nerve secured and divided. Effect of electrical stimulation and avulsion studied under screen examination.

*X ray.*—Phrenic-nerve stimulation produced sudden spasm of the diaphragm and fixation of the lower ribs. Excessive stimulation brought about a convulsive spasm of the thorax in addition. During mild stimulation the diaphragm was seen to be in spasm and fixed in full contraction, the lower rib interspaces were narrowed. The heart appeared to have deviated to the right. (Fig. 308.)

The phrenic nerve was avulsed, slowly and completely. The wound was closed with linen sutures. Subsequent radiological examination revealed cessation of movements and elevation of the right half of the diaphragm. The mediastinum was normal. (Fig. 309.)



*Group IX.*—Avulsion of phrenic nerve and production of paresis of one half of the diaphragm, followed by the bronchoscopic introduction of gum acacia.



FIG. 310.—*Group IX.* Complete density of lower half of the right lung. Displacement of the heart to the right. Intrabronchial gum acacia and phrenic neurectomy.

Dog, male, weight 9.4 kilos.

Anæsthesia: amytal 5 c.c.; morphine 1 gr.; open ether.

Avulsion of right phrenic nerve carried out as in previous experiment. Paresis of the right half of the diaphragm was confirmed by X-ray examination.

*One Month Later.* Anæsthesia: amytal and morphine.

Bronchoscope passed and 15 c.c. of 75 per cent gum acacia introduced to the lumen of the right bronchus, when it was occluded. Twenty minutes later some dyspnoea was observed, and on screen examination an increase in the density of the lung shadow of the affected side was noted.

In 24 hours: massive collapse of the right lung was observed, and the X-ray photograph was typical, with complete density throughout the right lung and deviation of the heart and mediastinum to the right. (*Fig. 310.*)

Resolution was complete in ten days. Massive collapse of the right lung had been produced.

The experiments were also carried out in animals on which the left side was similarly dealt with. Identical results were obtained.

### RESULTS OF EXPERIMENTS.

A solid foreign body placed in the lumen of the bronchus under narcosis was coughed up on recovery from the anæsthesia (*Group IV*). In *Groups III, V, and VI* areas of lobular collapse were found in the lung. In *Groups VII and IX* massive collapse of the lung was produced. In *Group VII* gum acacia (75 per cent), of viscosity similar to that of the intrabronchial secretion from a patient suffering from massive collapse of the lung, was introduced to the right bronchus of a narcotized dog. The procedure was followed by the application of strapping to the lower ribs of the animal. In *Group IX* a previously phrenicectomized animal was taken. Gum acacia of the same viscosity as in *Group VII* was introduced to the bronchus of the phrenicectomized side, under narcosis.

It will be seen that three factors, acting in combination, were necessary for the experimental production of massive collapse of the lung, namely: (1) *Intrabronchial content of definite viscosity*; (2) *Abolition of the cough reflex*; (3) *Limitation of respiratory movement*.

1. *Viscosity of Intrabronchial Content.*—It was found that the more fluid types of intrabronchial content, i.e., under 70 per cent gum acacia, were expressed proximally and expelled after the return of the cough reflex. No

interference with respiratory function occurred, and at most scattered areas of lobular collapse were seen in the radiogram of the chest. A solid foreign body plugging the bronchus was always expelled on the return of the cough reflex, after expulsion rapid aeration of the lung followed, and no areas of collapse were seen on the radiogram fifteen hours later. The means by which there was obtained a gum acacia solution of viscosity similar to the secretion in the human bronchus in a case of massive collapse, has been described. The estimation of 75 per cent gum acacia strength was arrived at by comparison in test-tubes. On studying the effect of the introduction of 75 per cent gum acacia with the bronchoscope, it is seen that the viscous material clings to the wall of the bronchus. There is a slight to-and-fro movement with inspiration and expiration. As the bronchus dilates with inspiration, however, the gum moves distally farther down the lumen. Soon, as the diameter of the lumen of the bronchus diminishes, partial and finally complete obstruction occurs. As one writer has put it: "the secretion agglutinates to obstruct".

**2. Abolition of the Cough Reflex.**—The use of morphine as an adjuvant to amytal anaesthesia was particularly valuable. The cough reflex remained inactive during the intrabronchial manipulations provided these were carried out gently and without trauma to the mucosa. At the end of the experiment the animal remained more or less asleep in its cage for an hour or two, and no effectual spasm of coughing took place. Later the animal moved about and drank water. Vomiting did not occur. If effectual coughing did take place following traumatization, the gum acacia was expelled, the air-passages were cleared, and the lung became fully aerated. Persistent deep narcosis was found to be unnecessary both for the intrabronchial or intra-abdominal manipulations, provided these were carried out gently.

**3. Limitation of Respiratory Movement.**—In *Group VII* this was produced by the application of adhesive strapping to the lowest ribs on one side. The tension was similar to that sometimes used when securing an upper abdominal dressing and less than that for fractured ribs. The strapping was applied over the hairy coat, to allow a fair margin of movement with respiration. In *Group IX* limitation of respiratory movement had been brought about some weeks previously by avulsion of the phrenic nerve on one side. The subsequent paralysis of one half of the diaphragm was always confirmed by radiographic examination. In phrenicectomized animals no further measures to limit respiratory movement were taken, and no strapping was applied to the chest after the introduction of the gum acacia to the bronchus. It was found that in these animals a paramedian epigastric laparotomy incision, made and closed under aseptic surgical conditions, produced no limitation of respiratory movement. Complete aeration of the lung bases was confirmed by radiographic examination. The animals moved about in their cages with apparent freedom on the night of operation, and in none of the experiments was laparotomy a causative factor in the production of massive collapse of the lung.

## APPLICATION OF EXPERIMENTAL FINDINGS TO SURGICAL PRACTICE.

Under this heading the question must be asked: Are these three factors —(1) viscid intrabronchial content, (2) abolition of cough reflex, (3) limitation

of respiratory movement—present in the post-operative period in surgical practice? If they are, then a certain incidence of post-operative collapse of the lung is to be expected.

**1. Viscid Intrabronchial Content.**—The 'wet' anæsthetic is too well known to warrant further description. The bronchial hypersecretion may have accompanied a temporary or chronic bronchial catarrh, or it may be due to the action of irritant anæsthetic vapour on a sensitive mucous membrane. Cigarette-smoking has been blamed as an etiological factor. Asthmatics, as a rule good subjects for careful inhalation anæsthesia, have also been mentioned. The frothy hypersecretion of the typical 'wet' anæsthesia is probably of less importance in the etiology of massive collapse than the more viscid type of secretion obtained in much smaller quantity from those patients in whom chronic irritation has produced a more patchy type of reaction in the respiratory passages. To this group smokers and asthmatics belong.

**2. Abolition of Cough Reflex.**—The cough reflex is abolished in every inhalation anæsthesia. In most cases operated on under local or spinal anæsthesia a preliminary sedative or narcotic is given. The intrabronchial content is subject mainly to the rhythmic movements of the bronchial tube alone. It is known that an intrabronchial content above a certain viscosity tends to be indrawn to the smaller branches of the bronchial tree. The right bronchus offers a more direct route to the descent of such retained secretion. In the immediate post-operative period it is customary to administer morphine or its derivatives, to allay pain. The sedative action of morphine on the respiratory centre and the cough reflex is well known. Later the pain in the wound produced by coughing is such that the conscious patient voluntarily tries to avoid any spasmodic respiratory effort such as coughing, sneezing, or laughing.

**3. Limitation of Respiratory Movement.**—Quiet regular breathing is demanded from his anæsthetist by every surgeon. No deficiency in aeration of the lung should occur, however, unless the presence of intra-abdominal packs, or the operative position of the patient, should unavoidably interfere to some extent with the descent of the diaphragm or rib excursion. In such cases the lung bases alone are deficiently aerated. The application of over-tight bandages, especially when such bandages extend over the costal margin, is a potent factor in limiting post-operative movement of the abdomen and ribs during respiration. The narcotized respiratory centre maintains breathing of a shallow type only. Aeration of the lung bases is considerably impaired. Secondary factors of posture and flatulent distension are also of importance. The supine patient does not have the aid of gravity in the descent of the liver during inspiration. Similarly the patient placed in an exaggerated Fowler's position, hunched forwards, has no facility to expand the lower ribs, and gravity brings the liver no farther than against the posterior abdominal wall.

The effect of operation on the vital capacity during the post-operative period was studied by Head,<sup>18</sup> whose results are :—

Operations on lower abdomen	..	..	30-40 per cent	dim. V.C.
„ „ upper abdomen	..	..	78-88 per cent	„ „
„ „ thorax wall	..	..	14-34 per cent	„ „

Particular stress was laid on the subsequent limitation in the descent of the diaphragm, and the frequent subsequent development of small scattered areas of lobular collapse in the lung. This inability of the normal post-operative patient completely to aerate his lungs is complementary to his fear of movement, and to his helplessness in the presence of pain, real or anticipated.

In our experience with the experimental animal the experiments involving introduction of a bronchial plug and laparotomy failed to produce collapse, largely owing to the tendency of the animal to struggle to its legs and move about in its cell immediately on waking up from its anæsthetic. The post-operative efficiency of aeration of the lungs must have been raised beyond comparison with that of the human subject.

In addition to involuntary splinting of the abdominal muscles after operation, the plain muscle of the intestine remains quiescent and peristalsis is in abeyance. The effect of flatulent distension in limiting diaphragmatic descent is well known. It is also likely, however, that some diminution of diaphragmatic activity is reflex, initiated from its association with peritoneum which has established a control of all movement likely to be noxious to itself or its contents.

### THE PATHOLOGICAL PROCESS.

A few pneumococci and certain streptococci may be isolated from the healthy human bronchus. Similarly in the healthy dog limited numbers of pneumococci and streptococci may be isolated from the bronchial mucosa. The bronchial secretion from a patient suffering from massive pulmonary atelectasis was found to be heavily infected with pneumococci, and when some of it was placed in the bronchus of a healthy animal, a rapidly fatal and widespread bronchopneumonia at once developed. When massive collapse is experimentally produced in an animal and the animal is killed within twenty-four hours, the appearances in the lung post mortem are entirely those of collapse. The lung is heavy and darkly congested; it is no longer crepitant. The size is only slightly less than normal. Microscopically the alveoli are found to be collapsed. The lining cells are œdematous, the vessels are congested, and many red blood-corpuscles are lying both in the alveoli and in the stroma (*see Fig. 306*). The exudation containing blood-cells is a physical reaction following the absorption of air from the alveoli and the subsequent diminution in the intra-alveolar pressure. When massive collapse has been of longer duration before death occurs, the appearances post mortem become those of bronchopneumonia. This is the result of infection from the retained bronchial secretion which forms a culture medium for the pneumococci originally present. The œdematous and collapsed peribronchial alveoli become heavily infected, and both bronchioles and alveoli are occupied by a purulent exudate. Death from massive collapse of the lung *per se* rarely occurs before the morbid appearances of bronchopneumonia have become established.

A complete recovery, clinical and physical, is the rule in the majority of these cases. Probably because of this, there has been a tendency to underestimate the importance of atelectasis as the origin of serious ill health in a minor group. The case mortality of a post-operative pulmonary complication

has been estimated at 20 per cent. If, however, cases are included which suffer from transient pulmonary symptoms only and in which the diagnosis of pulmonary collapse is only made on a presumptive clinical basis or after radiological examination, the total number of post-operative pulmonary complications will be considerably raised and the case mortality will fall. The pathological conditions found in the lung as late results of incompletely resolved atelectasis are bronchiectasis and pulmonary abscess. The clinical signs which interpret the development of these conditions in a case of unresolving massive collapse are very variable. The radiological appearances, however, show that the mediastinum and heart shadows do not return so completely to their normal position, and areas of increased density remain at the site of collapse.

### THE PREVENTION OF POST-OPERATIVE PULMONARY ATELECTASIS.

Having regard to the combination of factors which predispose to the experimental production of massive collapse, the prevention of this post-operative condition will probably be most successful where co-operative measures are undertaken by the members of a surgical unit. Where possible, bronchial catarrh and oral sepsis should be corrected prior to operation. The choice of anæsthetic is important. Pre-operative atropinization of the patient is very valuable, but the use of atropine combined with heroin or morphine has the dangers associated with depression of the respiratory centre. To calm a patient prior to operation and to allay mental stress without acting as a depressant, no drug has proved more useful clinically than nembutal, which has been found safe and efficient with spinal, local, or general anæsthesia. The prevention of asphyxia and the prevention of accumulations of mucus in the respiratory passages are obvious measures to which the anæsthetist attends. The anæsthetist also has added carbon-dioxide gas to his armamentarium, by the use of which the respirations may be kept at sufficient depth, and the lung bases are preserved in a fully aerated state. At the end of operations which, by their nature, have produced some limitation of respiratory movement, full aeration may be re-established by the judicious use of carbon dioxide in the anæsthetic vapour.

Dressings should be secured in such a manner that they in no way interfere with respiratory movement. Such bandages, etc., may be adjusted or loosened from time to time if any tightness is apparent. In the same way the position of the patient may be altered to ensure full diaphragmatic excursion. The firm canvas bed-rest is probably the most efficient means of procuring a comfortable reclining Fowler's position with maximum aeration of the lung bases.

Sedative drugs which have a strong affinity for the respiratory centre should be withheld until the patient has regained full consciousness. In the immediate post-operative period a spasm of coughing or the retching of post-anæsthetic sickness leads to the expulsion of retained intrabronchial secretion and thorough aeration of the bronchial tree. The early administration of morphine or heroin may permit such secretion to remain in the bronchus, agglutinating to obstruct.

The early recognition of flatulent distension and its treatment by procuring the passage of flatus will help to diminish the incidence of the delayed type of pulmonary lobular collapse.

## TREATMENT.

The treatment of cases of post-operative massive collapse of the lung was until recently the routine treatment administered to cases of respiratory infections as a whole. There were advised poultices to the chest wall, medical diathermy, expectorant mixtures, steam tents, various antisera and vaccines.

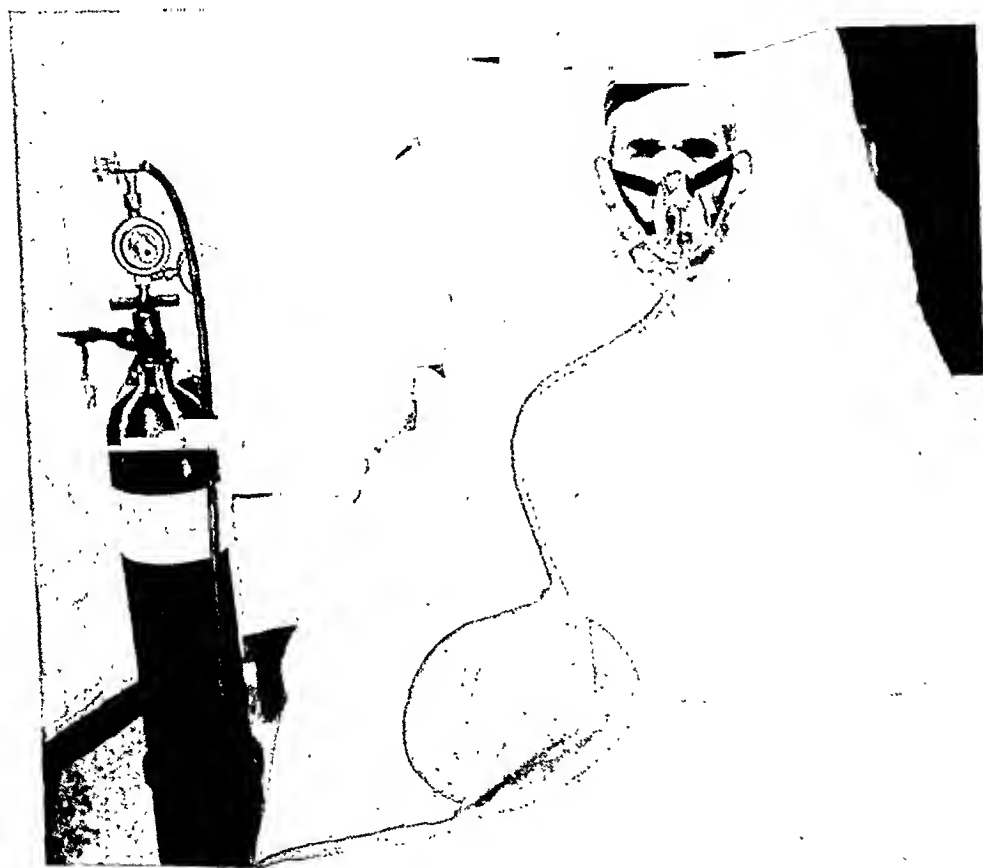


FIG. 311.—Carbon-dioxide therapy.  $\text{CO}_2$  is being administered through a Haldane's mask inhaler.

Undoubtedly these were all helpful measures, particularly when broncho-pneumonia had become established and the cough was productive of a purulent sputum. Associated infection of the pleura required symptomatic relief. Having regard, however, to one of the main factors in the etiology of massive collapse—shallow breathing—*carbon-dioxide gas becomes an important remedial measure.*

Carbon dioxide is supplied from a cylinder containing 10 per cent of the

gas in oxygen. The gas may be supplied direct to the patient from the cylinder through a length of tubing and a mask inhaler (*Fig. 311*), or a bag may be interposed to act as a reservoir. Carbon-dioxide-mixture inhalations are administered for ten minutes in every hour. The increased respiratory depth and the productive cough that follow give the patient very rapid relief. The frequent, short, non-productive coughs which interfere with sleep are abolished in the intervals between inhalations. On clinical examination of areas in the chest in which breath-sounds were absent on auscultation, vesicular breathing is found to have returned; the accompanying râles indicate the mobilization of secretion and the re-aeration of the alveoli. In early cases of pulmonary collapse complete recovery may be brought about in forty-eight to seventy-two hours. In more established cases there is complete symptomatic relief in that period, though radiological evidence of the condition is still present. In a small percentage of cases of post-operative massive collapse, in which the whole of one lung is affected, coughing is not sufficiently productive, and the sputum is small in quantity and difficult to bring up. In these cases bronchoscopic aspiration of the sticky mucus gives immediate relief, and the danger of a severe infection from the retained secretion will be avoided. Similarly, where radiological or clinical evidence of bronchiectasis is present in cases which fail to clear up completely after pulmonary atelectasis, bronchoscopic aspiration is a valuable aid to treatment.

### SUMMARY.

1. Records of four clinical cases of post-operative massive collapse of the lung are given.

2. Experimental evidence is brought forward indicating that certain factors, acting in combination, may lead to collapse. These are: (a) A viscid intrabronchial secretion (viscosity 75 per cent); (b) Abolition of the cough reflex; (c) Limitation of respiratory movement.

3. Preventive measures are suggested.

4. The therapeutic value of carbon-dioxide inhalations is emphasized.

We are grateful to Mr. Henry Wade and Dr. G. Ewart Martin for their interest in this investigation. We owe Professor D. P. D. Wilkie our sincere thanks for his guidance both in the experimental laboratory and in the wards, where access to clinical cases was so kindly given. We would like to mention the valued services rendered by Mr. F. W. Pettigrew and the technical staff of the Surgical Research Department, University of Edinburgh. The expenses of this research were covered by a grant from the Earl of Moray Fund, University of Edinburgh.

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## AN ANALYSIS OF 2126 CASES OF ACUTE APPENDICITIS.

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IN the following tables an analysis has been made of the results of treatment of acute appendicitis at St. Thomas's Hospital during the years 1920-9 inclusive. This is of particular interest, as a similar investigation<sup>1</sup> carried out for the years 1894-1903 inclusive allows a comparison to be made between present results and those of thirty years ago. For this purpose the same grouping of the cases has been adopted.

We have endeavoured to include only those cases in which definite acute inflammation of the appendix was found at operation, or—if no operation were undertaken—those in which the history and clinical examination left little doubt as to the diagnosis. The classification into the following groups was made on the pre-operative clinical signs, rather than on the operative findings, because we consider that the presence of a palpable mass has a bearing of the greatest importance upon the line of treatment.

*Table I* contains the cases of acute appendicitis admitted during the first four days of the disease in which the inflammatory process was limited to the appendix or a localized peritonitis had occurred.

It is an established fact that appendicectomy carried out during the first twenty-four hours from the onset of the disease carries a very low mortality-rate—in our series 0.22 per cent. On subsequent days it rises to a much higher figure, but in this group does not continue to rise after the first forty-eight hours. The probable reason for this is the varying rapidity in the progress of the disease. It is noticeable, however, that the complication-rate appears to rise steadily, in direct relation to the time which elapses before operation.

There were a few cases, diagnosed as acute appendicitis and admitted during the first four days, which were not operated upon. These showed a much higher mortality- and complication-rate, although they appeared to be suffering from milder attacks.

*Table II* contains the cases of appendicitis with diffuse peritonitis admitted during the first four days of the disease, and immediate operation with removal of the appendix would appear to be the only rational form of treatment to adopt. Four cases treated only by drainage died; of five cases in which no operation was undertaken, four died: the fifth was considered too ill for surgical treatment, but recovered.

If appendicectomy is carried out, drainage of the peritoneum seems of little importance.

It is interesting to note there is little relation between the duration of

Table I.—ACUTE APPENDICITIS WITHOUT DIFFUSE PERITONITIS (1408 CASES).

## A. Immediate Operation (1340 Cases).

DAY OF ILLNESS	NO. OF CASES	MORTALITY		COMPLICATIONS	
		Cases	Per cent	Cases	Per cent
1st .. .. .	442	1	0.22	7	1.58
2nd .. .. .	531	17	3.20	27	5.08
3rd .. .. .	293	3	1.02	34	11.60
4th .. .. .	74	2	2.70	6	8.10
Totals .. .	1340	23	1.71	74	5.50

## B. Analysis of Complications (74 Cases).

COMPLICATED BY	1ST DAY	2ND DAY	3RD DAY	4TH DAY
Residual abscess* .. ..	5	12	22	3
Subphrenic abscess .. ..	1	2	2	2
Obstruction .. .. .	1	3	3	—
General peritonitis .. ..	—	3	2	1
Pleural effusion .. .. .	—	1	1	—
Pulmonary embolism .. ..	—	1	—	—
Femoral thrombosis .. ..	—	3	—	—
Portal pyæmia .. .. .	—	2	1	—
Empyema .. .. .	—	—	1	—
Septicæmia .. .. .	—	—	1	—
Fæcal fistula .. .. .	—	—	1	—
Totals .. .. .	7	27	34	6

\* Note.—Residual abscess occurred in 4 cases in which a drain was used, and in 33 cases in which the wound was closed without drainage.

## C.—Summary of Cases of Acute Appendicitis admitted on First Four Days of Illness, which were not Operated upon (68 Cases).

DAY OF ILLNESS	DISCHARGED: NOTHING FURTHER KNOWN	DISCHARGED: REMOVAL LATER	WENT WRONG
1st .. .	0	1	0
2nd .. .	3	6	3 developed abscess
3rd .. .	7	11	5 developed abscess: 1 died, 2 had acute attack a month later
4th .. .	10	13	1 developed abscess; 1 died unoperated; 1 died after operation 2 days later; 6 developed acute attack, necessitating operation at intervals of 1, 3, 9, and 12 weeks respectively.
Totals	20	31	17

More than 25 per cent go wrong, and 4.4 per cent die.

the disease and the death-rate, which is as high on the first day as on any of the others.

*Table II.*—231 CASES ADMITTED WITH DIFFUSE PERITONITIS.

DAY OF ILLNESS	APPENDICECTOMY AND CLOSURE		APPENDICECTOMY AND DRAINAGE		DRAINAGE ONLY		NO OPERATION	
	Recovered	Died	Recovered	Died	Recovered	Died	Recovered	Died
1st .. ..	9	3	5	0	—	—	—	—
2nd .. ..	32	7	22	6	—	—	—	—
3rd .. ..	37	10	38	8	—	3	—	3
4th .. ..	17	5	18	5	—	1	1	1
Totals ..	95	25	83	19	—	4	1	4

MORTALITY { Appendicectomy and closure = 20·8 per cent  
 { Appendicectomy and drainage = 18·6 per cent.

*Complications in Diffuse Peritonitis Cases (37 Cases).*

COMPLICATED BY				2ND DAY	3RD DAY	4TH DAY
Fæcal fistula .. ..	..	..	..	1	1	—
Pleural effusion .. ..	..	..	..	1	—	—
Residual abscess .. ..	..	..	..	5	13	2
Pulmonary embolism .. ..	..	..	..	1	—	—
Subphrenic abscess .. ..	..	..	..	1	2	—
Femoral thrombosis .. ..	..	..	..	—	1	—
Obstruction .. ..	..	..	..	—	3	1
Empyema .. ..	..	..	..	—	2	1
Abortion .. ..	..	..	..	—	2	—
Totals .. ..	..	..	..	9	24	4

CONCLUSION.—In cases with diffuse peritonitis operated upon in the first four days, the death-rate is 20 per cent, and the percentage of complications is 20·6 per cent.

*Table III* contains the cases of appendicitis in which a localized mass could be felt at the time of admission. It is in this group that most discussion arises as to the correct treatment to be adopted. It has been the practice in this hospital to follow a conservative course where possible; but if the urgency of the symptoms makes operative interference essential, drainage alone is the method of choice. In some instances, however, where there is evidence of spreading infection of the peritoneal cavity, appendicectomy and drainage is performed, as this type of case is comparable to those in Group II.

Although this conservative method of treatment necessitates readmission for appendicectomy after a suitable interval, it is found in practice to involve little or no added risk. Of such cases, 299 were operated upon, with no deaths.

It is often stated that the adoption of any form of conservative treatment for acute appendicitis leads to an increase in the general mortality of

Table III.—487 CASES ADMITTED WITH PALPABLE MASS.

DAY OF ILLNESS	OPERATION					NO OPERATION				
	Appendicectomy and Drainage		Drainage Only			Subsided		Evacuated by Bowel		
	Dis-charged	Died	Dis-charged: Nothing further known	Returned for Appen-dicectomy	Died	Dis-charged: Nothing further known	Returned for Appen-dicectomy	Dis-charged: Nothing further known	Returned for Appen-dicectomy	Died
2nd ..	9	1	1	1	0	2	2	—	—	—
3rd ..	14	0	2	4	1	13	14	—	—	—
4th ..	10	4	3	8	1	17	27	—	—	1
5th ..	4	1	4	6	0	18	23	—	1	1
6th ..	6	2	3	13	1	15	17	—	—	—
7th ..	1	0	0	10	0	9	17	—	—	—
Over 7th	11	2	14	50	2	48	63	4	6	—
Totals	55	10	27	92	5	122	163	4	7	2

MORTALITY { Appendicectomy and drainage = 15.4 per cent  
 { Drainage only .. .. = 4.03 per cent } Mortality in subsequent  
 { No operation .. .. = 0.67 per cent } appendicectomy = Nil.

## Complications in Acute Cases admitted with Palpable Mass.

COMPLICATED BY	CASES OPERATED ON	CASES NOT OPERATED ON
Acute attack .. ..	7	11
Residual abscess .. ..	12	5
Obstruction .. ..	5	2
Pulmonary embolism .. ..	1	—
Faecal fistula .. ..	3	—
Subphrenic abscess .. ..	—	1
Femoral thrombosis.. ..	—	1
Totals .. ..	28*	20†

\* Out of a total of 161 cases operated on = 17.1 per cent.

† Out of a total of 296 cases not operated on = 6.76 per cent.

CONCLUSION.—In 'appendix mass' cases of all stages, cases operated upon give 7.9 per cent mortality and 17.1 per cent complications. Cases not operated upon give 0.68 per cent mortality and 6.76 per cent complications.

Table IV.—COMPARISON OF AN EARLY WITH A RECENT GROUP OF CASES OF ACUTE APPENDICITIS OPERATED UPON AT ST. THOMAS'S HOSPITAL.

Group A (1894-1903).—433 cases with 182 deaths (42 per cent).

Group B (1920-1929).—1755 cases with 86 deaths (4.9 per cent).

## I. EARLY ACUTE CASES :—

A.—13 with no death.

B.—1340 with 23 deaths (1.71 per cent).

## II. CASES WITH DIFFUSE PERITONITIS :—

A.—166 with 143 deaths (86.0 per cent).

B.—226 with 48 deaths (21.1 per cent).

## III. CASES WITH PALPABLE MASS :—

A.—254 with 39 deaths (15.3 per cent).

B.—189 with 15 deaths (7.95 per cent).

the disease. It is for this reason we wish to make it quite clear that we do not consider that every patient in whom an appendix mass can be felt is safe to be left. Careful observation and sound judgement are essential, for out of our 487 cases, 120 required immediate surgical treatment; and of those which appeared likely to subside when first seen, 69 were subsequently explored on account of failure to resolve, or exacerbation of symptoms.

### CONCLUSION.

1. All cases of acute appendicitis in which no mass is palpable require immediate surgical treatment.
2. In cases with diffuse peritonitis removal of the appendix is essential.
3. If a palpable mass is present the best results are obtained by conservative treatment, where this is possible.

We are indebted to the surgical staff of St. Thomas's Hospital for permission to publish their cases, and especially to Sir Percy Sargent, who is chiefly responsible for the arrangement of the figures.

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## THE RADIUM TREATMENT OF CARCINOMA OF THE BREAST.

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### THE RADIUM PROBLEM.

THE radium problem in the treatment of carcinoma of the breast is exactly the same as that which faces purely operative treatment—how to extirpate completely the cells of the primary growth and all secondary growths that may conceivably be accessible. Advance in the surgery of the breast dates from 1867, when Charles Moore,<sup>1</sup> of the Middlesex Hospital, criticized the methods of removal of the breast then practised. He advocated the wide removal of skin and underlying tissues, and of the main lymphatic glands draining the breast. He also enunciated the principle of the centrifugal dispersion of the disease. Since his time the work of Mitchell Banks, Samuel Gross, Halsted, Sampson Handley, and Cheate (to mention only some of the outstanding names) has fully established the correct principles of surgical treatment, and the operation has been virtually standardized in the removal of: (1) The whole mammary gland with the overlying skin for as great a distance as possible around the primary growth; (2) The underlying fascia and pectoral muscles over a considerably greater area; and (3) The axillary lymph glands and channels from the breast in continuity. Attempts were made by Halsted to extend the operation still further by dividing the clavicle and carrying the dissection up into the supraclavicular region, and Harvey Cushing, working in Halsted's clinic, removed on several occasions the lymphatic glands in the anterior mediastinum. These extensions have not become standardized, chiefly owing to their technical difficulty and the danger to the patient, though also, perhaps, because the results did not justify so difficult and dangerous a procedure. Without them, the operative mortality for cancer of the breast has been extremely low, and, owing to the relative accessibility of mammary cancer, the results, compared with those obtained in other parts of the body, have been good. At the same time there has always been a fairly rigid segregation of the patients into surgical sheep and goats—the two classes known colloquially as 'operable' and 'inoperable'—and in every clinic in this country, as in others, the latter category has been lamentably large. The label 'inoperable' has depended on somewhat gross criteria—obvious local extension of the growth beyond the limits of operation, adhesion of the growth to the chest wall, involvement of lymphatic glands other than those in the axilla, or the presence of metastases in other parts of the body.

The radium problem may therefore be resolved into a series of questions which it is my object in this paper to make some attempt at answering:—

1. Can the primary growth in the breast be eradicated by radium?

2. Can secondary growths in lymphatic glands be eradicated, and what limits must be set to the treatment?

3. Can radium be used to cure or alleviate the disease in those patients who are already from the surgical point of view 'inoperable'?

4. Are the final results to be obtained by radium or by radium combined with surgery in 'operable' cases as good as, worse than, or better than those obtained by surgery alone?

In attempting to answer these questions I shall assume that no miracles can be performed by radium. If the patient already has metastases in other parts of the body, no form of local treatment, whether operative or radiological, will save her life. At the same time it is clear that there is a stage in every case of carcinoma of the breast during which the disease is a local lesion, and therefore any form of local treatment must depend for its success primarily on early diagnosis. This will apply as much to radium as to operation, and since the 'clinical material' at my disposal has been entirely unselected, and for the most part extremely unfavourable, clearly no startling end-results can be expected when they are expressed statistically. The study of individual patients will throw more light than any figures, and case-histories will be an important feature of this contribution to the solution of the radium problem.

### THE HISTORY OF RADIUM TREATMENT OF CARCINOMA OF THE BREAST.

The history of the radium treatment of carcinoma of the breast is largely contained within the pages of this article. Until comparatively recent times radiological treatment was reserved for those patients who were clinically 'hopeless'—those who had advanced and inoperable tumours, or were surgical failures, having developed external recurrences after operation. Considerable numbers of such patients had been treated by external application of radium. Thus at the London Radium Institute<sup>2</sup> no fewer than 633 patients had received this form of treatment in the period 1918–27, but only 164 of these had primary inoperable tumours, the remainder having post-operative recurrences. The results obtained were encouraging, 26 out of 128 patients surviving for three years, and 11 out of 98 patients surviving for five years; but, in spite of this, no advance was made towards treating patients in a more hopeful stage of the disease. In Continental radiological clinics similar classes of cases have been treated, but it has been for the most part assumed that nothing further was possible, and, indeed, that it would be wrong to attempt the treatment of operable tumours with radium. One of the leading Continental authorities, when told recently of work being done in London on newer lines, showed but little interest, and dismissed the subject with the laconic remark, "He will be disappointed."

In treating carcinoma of the breast with radium it is at once obvious that two main methods are available: (1) External irradiation with a comparatively large quantity of radium; and (2) Interstitial irradiation with a smaller quantity. When in 1922 radium was put into the hands of the Surgical Professorial Unit at St. Bartholomew's Hospital under the direction of Professor G. E. Gask, the amount available was small, and interstitial irradiation was

clearly the method to be chosen, the details of procedure being decided upon in consultation with Dr. N. S. Finzi. The radium was distributed in small units, 1 mgrm. to 1.6 cm. in platinum needles with walls 0.5 mm. thick, and a cautious beginning was made by treating recurrent nodules of growth following radical surgical removal of the breast. It had already been ascertained that interstitial irradiation with a comparatively large quantity of radium, such as a tube containing 50 mgrm., for a short period was apt to prove too intense and to result in damage to normal tissues with necrosis. It was resolved, therefore, from the start of this investigation, to use only the small units, the largest needle containing 3 mgrm. of radium element in an active length of 4.8 cm., and to prolong the time of irradiation by several days.

During the years 1922-4 seven patients with recurrences were treated, and in almost every instance complete disappearance of the growth resulted. One of these patients is alive and well at the present time, nearly eight years after treatment, but the others all died within a comparatively short time from metastases in other parts of the body. Although their lives had not been prolonged, valuable information had been obtained from these preliminary experiments. It was clear that a nodule of recurrent growth, whether surgically removable or not, could be made to disappear almost with certainty, and if this could be done with a recurrent growth, why not also with a primary growth? The conclusion appeared to me to be that primary carcinoma of the breast might be successfully treated in the same way, and with Professor Gask's concurrence it was decided to attempt this, the experiment being limited to patients whose tumours were regarded as inoperable according to the ordinary standards.

The first patient to be treated in this way was a woman aged 47 with a small carcinoma of the left breast which had been present for three years and had become adherent to the chest wall, so that it was inoperable. The treatment was given on Aug. 1, 1924, and the full case-history is as follows:—

*Case 1.* Age 47. Referred to the Surgical Unit as inoperable by Mr. H. W. Wilson. A small lump had been noticed at the upper and inner quadrant of the left breast for three years. On examination the patient was found to have a hard contracting tumour, 3 × 4 cm., which was infiltrating the skin, ulcerated in the centre, and adherent to the pectoral muscle. Radium treatment was given on Aug. 19, 1924, 52.8 mgrm. of radium element being inserted deep to the growth for 24 hours. A month later the ulcer, present for a year, had healed. The whole mass was smaller and more mobile, and the patient had gained 5 lb. in weight in three weeks. A second treatment was given on Dec. 4, 11.5 mgrm. being inserted for 120 hours. Further improvement resulted, and the breast began to fall towards its normal position. By July 2, 1925, the growth had disappeared except for some small nodules in the skin, and this remaining portion of the growth was excised. The presence of carcinoma in the nodules was confirmed by section. On Jan. 18, 1927, the patient had no local recurrence, but there was a palpable gland in the left axilla, and the lymph areas were then treated in the ordinary way. The axillary gland had disappeared by April 4, 1927. The patient remained well until Nov. 5, 1929, when a nodule of growth was found in the skin of the areola—that is, outside the area originally treated. A fourth treatment was given for this on Jan. 10, 1930. The patient is alive without signs of active disease at the present time (7 years after the initial treatment). (*Figs. 312, 313.*)

This patient suffered by reason of having come before the technique had been developed, so that she had to be treated in four stages. Ultimately,



however, the full treatment was given, and the patient, who started with an inoperable growth seven years ago, is quite well at the present time.

In the first two patients treated there happened to be no palpable lymphatic glands, and treatment was given only to the primary growth. As soon as a patient with palpable lymph glands was to be treated, the question arose whether the lymphatic areas were to be treated at the same time as the primary growth. It was decided to insert needles in the axilla and below the clavicle, and to do this as a routine whether glands were palpable or not. The effect on lymphatic glands already enlarged was found to be remarkably good, and as a prophylactic measure the treatment also seemed to be successful.

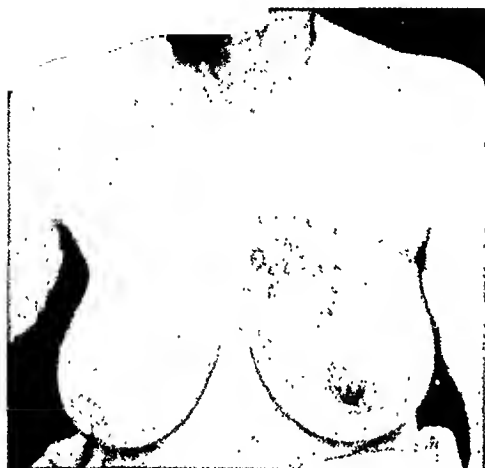


FIG. 312.—Case 1. Before excision of scar.

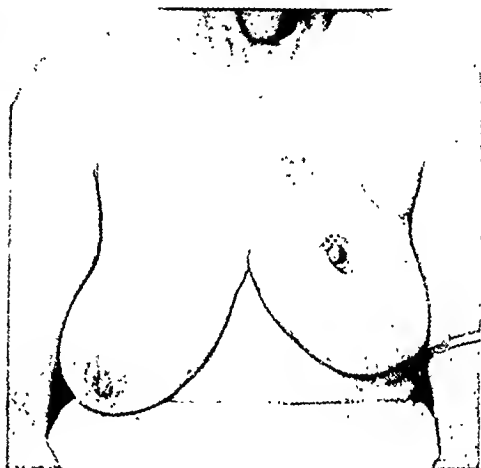


FIG. 313.—Case 1. Seven years after treatment.

In June, 1925, the routine treatment was extended to the supraclavicular glands, and soon afterwards needles were usually inserted into the upper three or four intercostal spaces with the object of irradiating the internal mammary lymph glands.

The results obtained in inoperable tumours during 1925 were so favourable that in 1926 I urged that the treatment should be extended to operable tumours, and with Professor Gask's approval the first operable growth was treated with radium on March 23, 1926.

The routine treatment was now fully established, and with minor variations has been since carried out by the Surgical Unit at St. Bartholomew's, by myself at St. Bartholomew's, at the Mount Vernon Hospital, Northwood, and on private patients.

More recently primary tumours, chiefly inoperable, have been treated with radium by Stanford Cade and E. Roek Carling at the Westminster Hospital. Small numbers have also been treated by others elsewhere, but very little on the subject has yet been published in confirmation or the reverse of my results. The technique used at the Westminster Hospital was essentially different from mine, being done chiefly with 1-mgrm. needles inserted at right angles to the skin, and the interstitial irradiation has been

supplemented with superficial irradiation through Columbia paste as described by Stanford Cade.<sup>3</sup>

From the beginning of this investigation the 'Follow-up Department' at St. Bartholomew's Hospital under Miss Ball has been an exceedingly important factor in carrying on the work. Miss Ball's skill in keeping in touch with patients after their initial treatment has contributed largely to the value of the results that have been obtained, and her work must take its place in the history of the opening stages of the radium treatment of carcinoma of the breast.

## TECHNIQUE.

At the present time the technique involves the treatment of two main areas: (1) The breast and primary growth; (2) The accessible lymphatic areas. This separation is made owing to the fact that whereas the lymphatic areas require an almost constant amount of radium, the amount required for the primary growth is very variable. Two or three times as much radium may be needed for a large breast in a stout patient as for a small contracting growth in the breast of a thin patient. It is therefore impossible to lay down rules for dosage. Any statement that so many milligramme-hours is the correct standard would be entirely misleading with so variable a factor in the calculation. The only rough guide that can be given

is the empirical observation that the best results seem to be obtained in the breast by using needles of 3 mgrm. in an active length of 4.8 cm. with a filtration of 0.5 or 0.6 mm. platinum, placed about 1.5 cm. apart (Fig. 314). That is to say, for an area of breast  $4.8 \times 4.5$  cm. (21.6 sq. cm.), 9 mgrm. of radium will be required, or approximately 1 mgrm. for each 2.5 sq. cm. of breast. This calculation takes no account of the thickness of the tissue to be treated, and sometimes in a stout patient additional needles have to be inserted in another plane, nearer the surface. Experience is the best guide in this matter, for an overdose may result in very unpleasant consequences for the patient. It

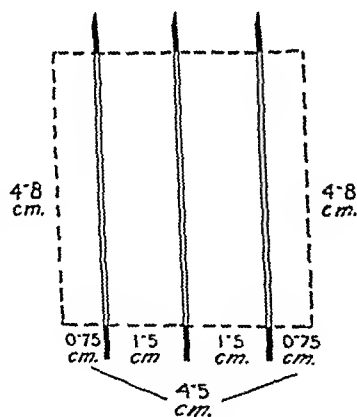


FIG. 314.

has been my practice to insert the needles as far as possible in a plane deep to the growth, usually in fact under the deep surface of the mammary gland itself—that is, in the plane of the pectoral fascia. My reasons for this have been several:—

1. Pushing needles into the growth itself, or even its immediate surroundings, has appeared to me to involve the risk of transplanting carcinoma cells into tissue previously uninfected. The ends of the needles are very feebly radio-active, so that any cells carried on the point will not be effectively irradiated.

2. Needles introduced into the sometimes poorly nourished tumour tissue may exert too intense a local effect, with risk of subsequent necrosis. Radium treatment can usually be made effective without necrosis and its attendant discomforts to the patient.

3. The main lymph channels draining the breast run in this plane deep to the breast.

4. The skin, being very susceptible to burning by radium, is liable to be injured unless screened by the breast tissue from the rays.

5. The breast tissue is often extremely tough, and needles may easily be damaged if any force has to be used while inserting them.

6. Experience has shown that when the mammary gland is not too thick an entirely satisfactory result can be obtained by this deep irradiation, sometimes without skin-burning or other disadvantage.

When the tumour or the breast is large I have sometimes departed from this principle by inserting additional needles more superficially, though these are usually in the neighbourhood of the growth rather than in it.

The needles are disposed as evenly as possible, sometimes in a cartwheel fashion, sometimes covering a quadrangular area. In the past the error has occasionally been made of covering an insufficient area of breast surrounding the primary tumour. Sir Lenthal Cheate<sup>4</sup> has recently stressed the fact that there are often many 'secondary deposits' in the breast itself outside the primary tumour; and therefore ideally the whole mammary gland should be irradiated in every case, and thus recurrences in the breast outside the area treated should be avoided. It is particularly important not to leave a gap of untreated breast between the tumour and the anterior fold of the axilla. This point will be further discussed in the section dealing with causes of failure (p. 451).

Sometimes the series of needles is continued under an area of skin in the neighbourhood of the breast, particularly towards the mid-line, if there is any suspicion of nodules spreading there. A much larger area than that actually occupied by palpable nodules must be irradiated. (*Fig. 315.*)

The accessible lymphatic areas are all treated in accordance with the known channels of lymphatic drainage of the breast. As demonstrated by Sappcy in 1885, the largest channels run round the outer border of the pectoralis major muscle—that is, in the anterior axillary fold—towards the axilla. It is a matter of common observation that in the great majority of patients the axillary glands are the group to be first infected with secondary growths, and it is reasonable to infer that carcinoma cells (whether they spread by multiplication in continuity, by active movement, or by embolism) take the line of least resistance—that is, towards the axilla in the first place. The treatment of the subpectoral lymphatics and of the axilla is therefore of the first importance and must never be omitted. This is best effected by the long needles (3 mgrm. in 4.8 cm. active length) inserted beneath the pectoral muscle, and, in continuity, into the axilla. Those in the axilla are placed on each wall in turn, four or five being placed so as to converge towards the apex. These can be placed with great accuracy through punctures in the skin, though care must be exercised not to pierce the pleural cavity by the needle on the inner and posterior wall, and not to injure vessels or nerves by that on the outer wall. The latter structures are safeguarded by keeping the upper arm abducted at a right angle from the body and inserting the needle in a direction parallel to the position of the neurovascular bundle and not too close to it.

The third lymphatic area for needles is the infraclavicular. It is not uncommon, when operating for carcinoma of the breast, to find enlarged glands running up from the axillary group over the costocoracoid membrane to a position almost behind the clavicle. Needles are therefore inserted below and more or less parallel to the middle third of the clavicle. Except in very stout patients, there is not room for the long needles, and it is better to use two or three needles each containing 2 mgrm. in an active length of 3.2 cm. These are inserted through skin punctures and are directed towards the axilla, but are plunged at first almost directly backwards so as to pierce the pectoral muscles. Their points should lie close to those of the needles in the axilla so that they may contribute to the irradiation of the apex of the axilla. Their radiations will also pass backwards to the costocoracoid membrane and upwards behind the clavicle.

The fourth lymphatic area is the supraclavicular. The glands in this area are commonly the next ones to become enlarged after the axillary glands. Occasionally they are affected before anything is palpable in the axilla, suggesting that there are some lymphatic channels passing directly upwards from the breast to the supraclavicular region without visiting glands in the axilla. Sometimes enlargement of these glands is undoubtedly due to an extension upwards of the

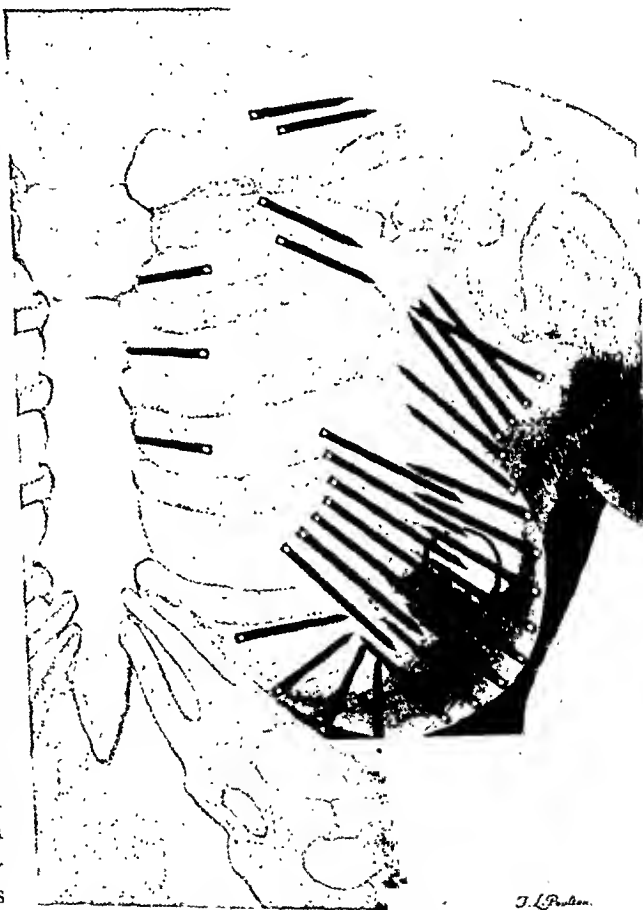


FIG. 315.—Diagram showing usual distribution of needles.

disease from the mediastinum, in which case radium treatment is unlikely to be of any avail; but it is seldom possible to be certain of this, and the supraclavicular group is certainly to be included in the routine treatment, thus extending the treatment beyond what is usually attempted by surgery, in spite of Halsted's earlier attempt to remove them. The needles, usually two or three of the shorter kind, are inserted through skin punctures from within outwards, parallel to the middle third of the clavicle. They should be plunged at first backwards through the deep cervical fascia so as to pass behind the posterior part of the sternomastoid muscle and the external

jugular vein. They then enter the loose areolar tissue at the root of the neck, and no force should be necessary to get them into position. If they are passed too deeply backwards, they will injure the brachial plexus, or if too much downwards may pierce the subclavian vein. If no force is used, neither of these accidents will occur.

The fifth lymphatic area includes the upper four intercostal spaces. During the 'following-up' of several hundred patients treated for carcinoma of the breast by surgery or radium in the course of the last eleven years I have never seen an intercostal recurrence traceable to one of the internal mammary lymphatic glands. Yet great stress has been laid upon the possibility of this event by Sampson Handley<sup>6</sup>, and I have followed his lead during the last four years by inserting radium needles into at least the upper three intercostal spaces in most patients. It is very dangerous to use long needles in this position, and the skin punctures must be made at a sufficient distance from the edge of the sternum. A needle of 2 mgrm. in 3.2 cm. active length is then pushed in very obliquely between the ribs, and the point must be felt to impinge on the lateral border of the sternum. The needle is then partly withdrawn and pushed in again slightly deeper so as to pass just behind the edge of the sternum. It should not project more than 1 cm. into the anterior mediastinum. If the needle is kept in this way very close to the sternum, it will not be in danger of injuring the pleura, pericardium, or internal mammary artery, though it will be in close proximity to the internal mammary gland. Any misjudging of the position of these needles may result in the loss of the patient's life.

A distribution of needles as described above will require up to about 100 mgrm. of radium element distributed as follows:—

10 needles	2 mgrm.	Ra each,	3.2 cm.	active length
25	„	3	„	4.8 „

The thickness of the platinum cases should be at least 0.5 mm., and it is probably better to have 0.6 mm. or even 0.8 mm. With the greater thickness virtually the whole of the  $\beta$  radiation is removed, so that the needles may be left in position for longer periods with less fear of damage to normal tissues. It is also a considerable gain to have the additional strength of the thicker needle when the tissues are tough.

Each needle is threaded on a strand of thick salmon gut, which has a single knot about 2 cm. from the eye of the needle. If the knot is close to the eye, the extraction of the needle is rendered more difficult. Salmon gut is used in preference to any form of thread, because it is non-absorbent, so that the punctures in the skin remain more completely aseptic than if silk is used, whether carbolized or plain. The salmon gut does not crack if it is kept moist for twenty-four hours before use. The strands of salmon gut are knotted together in groups of four or five, thus guarding against loss of any one needle should it be accidentally pulled out of the tissues. The knotted strands are wrapped in gauze to prevent irritation of the skin, and the whole area is covered with adhesive rubber strapping as a further safeguard against displacement or loss of needles.

The needles are usually left in position for a period of 7 days (168 hours). It has been found empirically that this period of irradiation gives satisfactory

## RADIUM TREATMENT OF BREAST CANCER 423

results with the dosage employed, and from the point of view of hospital organization a 7-day period is the most convenient arrangement. It has also been found that this exposure very seldom produces necrosis of the tumour. The skin not infrequently shows a superficial burn, which is satisfactory evidence that the irradiation has penetrated the tissues. Occasionally with a stout patient it may be advantageous to increase the period to 8, 9, or even 10 days, but it should scarcely ever be reduced to a shorter period than 7 days.

While the needles are in position the patient should be kept in bed and the arm on the affected side should be at rest, though it need not necessarily be kept in a sling. Most patients suffer but little pain except on movement; some of them complain of nausea while being irradiated, or may even vomit occasionally.

The needles are usually inserted under gas and oxygen anaesthesia. The operation is brief, and the patient suffers the minimum of disturbance with this anaesthetic. Removal may be effected in the same way, though a general anaesthetic is not an absolute necessity. Usually most of the needles can be extracted without any anaesthetic at all, and for those that are caught up in the subcutaneous tissue a few minims of novocain solution injected round the skin puncture are all that is needed.

After removal of the needles the punctures heal very rapidly and leave but little trace, though usually there is just enough scar to serve as a useful indication of the precise area that has been irradiated. Should the skin reaction be excessive, the peeled area will have to be dressed for several weeks, liquid paraffin or some neutral application being the best.

### AFTER-CARE.

As I have already mentioned above, the routine examination of the patients at intervals after treatment is an absolutely necessary part of the work—not only so that the surgeon may obtain information as to the efficiency of the treatment, but also because, in treating unselected cases, a single radium treatment must often be followed by further treatment, radiological or surgical.

During the present investigation I have usually seen the patients at monthly intervals after treatment for three to five months, then at three-monthly intervals for a year or more, and the interval has afterwards been lengthened to six months or longer, as time passed and confidence increased. Patients are also instructed to report themselves without being summoned if anything suspicious has been noticed.

When the needles are removed there is usually little change to be seen in the tumour or glands. Within a fortnight, however, there is often an appreciable alteration in size. As the weeks pass this becomes progressively more obvious, and sometimes, if the tumour was not large, or was particularly radio-sensitive, it may have disappeared completely at the end of five or six weeks after irradiation. A larger or less sensitive tumour will go on shrinking for a much longer period, and it may be three, four, or even five months before the full effect has been obtained. For this reason I have always been slow to apply further treatment, surgical or radiological, and in addition there is

nearly always a long latent period after treatment during which no renewed activity will appear even if the carcinoma is not completely destroyed. A rapid recurrence in the breast was seen in one patient who had had an inadequate treatment in the first place, but this is very exceptional (*Case 50*).

One factor in the problem is liable to produce an unfortunate uncertainty in the surgeon's mind, and that is the unknown quantity of fibrous connective tissue in the tumour and its surroundings. It was formerly supposed that one of the effects of exposure to radium was the formation of fibrous tissue, but possibly this was due to the caustic effect of the  $\beta$  rays, the amount of radium having been greater and the filtration less than in recent times. Certainly 'fibrosis' has not been an obvious feature of my treatment of carcinoma of the breast. On many occasions, indeed, the breast has become so soft and approximated so nearly to the normal that it has seemed that fibrous tissue has disappeared rather than formed.

*Case 68.*—Age 52. Lump noticed in right breast for more than a year. On examination there was a hard indefinite mass under the areola, 6 cm. in diameter. Nipple retracted. Skin not attached. Several hard enlarged glands in right axilla. Operable. Radium treatment on June 12, 1929. By Dec. 4 there was no discoverable sign of disease. The breast was quite soft and apparently normal. The patient has remained well since (2 years, 3 months).

*Case 73.*—Age 44. Lump noticed in right breast for several months. On examination there was an indefinite flat mass, 5 cm. in diameter, in the upper inner quadrant, adherent to the skin. No palpable glands. Operable. Radium treatment on June 26, 1929. By Aug. 28 slight thickening only was detected. At the present time the breast is quite soft and apparently normal (2 years, 2 months).

*Case 83.*—Age 58. Lump noticed in left breast for two years. On examination there was a very hard lump in the axillary tail of the breast,  $2.5 \times 2$  cm. Hard enlarged gland, 2 cm. in diameter, above this. Radium treatment on Sept. 11, 1929. By Nov. 21 both lumps had completely disappeared, and the breast has been apparently quite normal since that time (2 years).

On the other hand, many tumours undoubtedly contain a large amount of fibrous tissue which remains after the disappearance of the malignant cells, so that there is a residual tumour even though the treatment has been completely successful. Sometimes, also, the fatty tissue of the breast becomes denser and firmer after treatment, and this may also result in an apparent residual tumour. Very frequently the tumour disappears completely but the portion of the breast affected remains slightly thicker and firmer than the rest. This need not give rise to anxiety; but whenever there is an undoubted residual tumour I have always thought it wiser to give a second treatment or to remove a portion or the whole of the breast; occasionally I have done the removal after a second treatment. In some of the specimens thus obtained no residual carcinoma has been found; a fuller account of this will be found in a later section.

Another phenomenon which sometimes follows treatment is œdema of the skin overlying the breast. In several instances the breast underneath has been quite soft and has remained so; the œdema therefore may be due to lymphatic obliteration and not to recurrent growth.

*Case 65.*—Age 33. Lump noticed in left breast for five weeks. On examination there was a swelling in the upper outer quadrant, 2.5 cm. in diameter, not attached

to skin or deep fascia. Slight retraction of nipple. Large gland palpable in left axilla. Radium treatment on May 31, 1929. By Oct. 8 no tumour could be felt and no glands were palpable. On July 21, 1930, some oedema of the skin on the outer side of the breast was noted. A second radium treatment was given on Sept. 23. The oedema of the skin somewhat increased, but no tumour could be detected. The patient, however, developed signs of pulmonary metastases, and died on June 30, 1931.

## DIAGNOSIS.

It was clear from the outset of this investigation that it was necessary to set beyond doubt the nature of the disease that was being treated. I determined, therefore, to remove a specimen for microscopic examination from every patient, and this was done systematically in the first fifty patients treated. The specimen was taken at the time of *removal* of the needles—that is to say, after the growth had been irradiated for seven days or more. There are obvious theoretical objections to the removal of this specimen, such as the possibility of disseminating the growth by blood-vessels or lymphatics, or the encouragement of local growth. It was hoped to avoid these disasters by removing the specimen only after irradiation, with the idea that the growth of the malignant cells would have been inhibited, although their histological characters appeared to be unchanged. In this hope I was deceived, although the procedure afforded interesting information as to the biological behaviour of irradiated malignant cells. For in two patients an implantation growth appeared in the scar of the skin incision through which the specimen had been removed, although the primary tumour disappeared as the result of the treatment. In both of these patients further treatment was necessary, and in one of them the interference with the growth proved to be disastrous.

*Case 26.*—Age 39. Lump noticed in left breast for nine months. On examination there was a hard irregular lump in the upper part of the breast, 5 × 4 cm. in size, 2.5 cm. thick, attached to the skin. No glands palpable. Radium treatment on Dec. 16, 1927. Section removed on Dec. 23 showed spheroidal-celled carcinoma. On March 12, 1928, the original tumour had disappeared, but there was a small hard mass immediately under the scar of the biopsy incision. A second treatment was given to this on April 12, and by July 3 the swelling had disappeared. The patient remained well until Nov. 28, 1929, when a third treatment was given for secondary gland in the axilla. The patient subsequently developed signs of pulmonary metastases, though she is still alive at the present time.

*Case 32.*—This case is described on p. 448 under a different heading.

It appears, therefore, that an irradiated cell, though destined to disappear if left in its original site, may survive and multiply if transplanted to new surroundings. This observation may throw some light on the question of the effect of irradiation on malignant growths, whether it destroys the malignant cells, stimulates the other tissues to absorb them, or interferes with their blood-supply.

The observation was, indeed, too disturbingly interesting from the point of view of the patient's welfare, and I decided that the experiment was more suitable for performance on laboratory animals than on human beings. I considered, also, that histological proof of the nature of the disease in fifty consecutive cases was sufficient evidence in establishing the clinical results of radium treatment, especially as it had been obtained in some crucial cases,



notably the one described below under the heading of CLINICAL EVIDENCE. The histological proof has been obtained in a few later cases, when a local excision has been done before or after irradiation; but the diagnosis in the last hundred patients has depended for the most part on clinical evidence alone. The clinical diagnosis is, indeed, all too clear in most instances, and it is to be regretted that surgical teaching is still usually directed to emphasizing the late evidences of carcinoma of the breast rather than the early. When the diagnosis is clear the disease is usually past the earliest stage, and a number of patients in the present series who have been examined by their doctors during the early stage have been told to wait and so have only come for treatment when the diagnosis was obvious.

Carcinoma of the breast is so common a disease that suspicion should constantly be on the alert to detect it before puckering of the skin and other signs can be detected. Cysts of the breast, often difficult to distinguish by the fingers only, can always be excluded by the use of the exploratory syringe. The transillumination method of diagnosis, described by Max Cutler,<sup>6</sup> is of limited application, as the light cannot always be made to lie behind the growth unless the breast is somewhat pendulous, and I have been disappointed in its use. A fibro-adenoma usually presents the feature of mobility in the mammary gland, but I have on several occasions seen an equally mobile solid tumour which proved to be a nodule of carcinoma, and it is certain that every such tumour in a patient over the age of 25 should be regarded with suspicion. Another prevalent source of confusion is the condition designated 'mastitis', which may be closely simulated by a carcinoma growing somewhat diffusely from the start. Here the consistency, usually rather hard, should give the clue before there are any of the more obvious signs to be detected. I believe that an experienced clinician will make very few mistakes, and that in any case it is better to err on the side of diagnosing carcinoma too often than to miss it in the early stage. I think it is unlikely that many mistakes have been made in the present series. An exact record of the clinical findings has always been made in writing and is available for reference. Those patients in whom I felt there was genuine doubt as to the diagnosis have not been included in my records, so that if I have made an error it cannot amount to more than 1 or 2 per cent.

### EFFECT OF IRRADIATION ON TUMOURS.

It is necessary to describe in some detail the effect of interstitial irradiation given by this technique, and to take separately the different forms of growth. As it has not been my practice in recent years to obtain a section of the growth as a routine, it is not possible to 'grade' the tumours on any scientific basis such as Broders' classification. They will therefore be roughly classified on a clinical basis as 'early', 'intermediate', and 'advanced'.

1. **Early Growths.**—An early growth is a thing somewhat difficult to define, the patient's statement that it has only just been discovered so often being no proof that it is in an early stage. In general an early growth may be regarded as one which is small, which shows little sign of contraction or adhesion to neighbouring structures, and which is not associated with enlarged

## RADIUM TREATMENT OF BREAST CANCER 427

lymph glands. It is rare to find this type of growth among hospital patients, but much more frequent in private practice—that is, among the more educated classes. The effect of irradiation, as would be expected, is most rapidly apparent with the smallest tumours, even though they were of stony-hard consistency. Sometimes the tumour has completely disappeared within five weeks of removing the radium, and in such cases no recurrence has so far taken place. I have been able to place only 14 patients in the 'early' class, and in 7 the results are as detailed below. In the remaining 7 patients the carcinoma was excised locally before radium treatment was given, and these cases are described on p. 456.

*Case 59.*—Age 63. Lump noticed in right breast for two weeks. On examination there was a firm flat swelling, 2.5 cm. in diameter, in the inner upper quadrant. Skin slightly attached. No enlarged glands felt. Radium treatment given on April 9, 1929. On May 15 the tumour had completely disappeared, and the patient has remained quite well since (2 years, 5 months).

*Case 66.*—Age 57. Lump noticed in left breast for fifteen days. On examination in the inner upper quadrant was a firm rounded lump, 1.5 cm. in diameter, not adherent to the skin. Proved by aspiration not to be cystic. No glands palpable. Radium treatment given on June 8, 1929. By Sept. 20 the lump had completely gone, and the patient has remained well since (2 years, 3 months).

*Case 98.*—Age 73. Lump noticed in right breast for ten days. On examination a lump of 4 cm. in diameter was felt beneath nipple, which was retracted. No glands detected. Radium treatment given on April 9, 1930. One year later the patient was without signs of disease (1 year, 5 months).

*Case 99.*—Age 48. In the lower outer quadrant of the right breast was a hard lump, 4 × 2 cm., slightly attached to the skin. No glands palpable. Radium treatment given on March 21, 1930. The lump had disappeared two months later, and the patient has remained well since (1 year, 7 months).

*Case 105.*—Age 60. Lump noticed in right breast for one week. On examination there was a very hard lump, 2 cm. in diameter, in the upper outer quadrant, slightly attached to the skin. Small gland palpable in axilla. Radium treatment given on March 1, 1930. Patient next seen on Oct. 19 and found to be without sign of disease, and she has remained quite well since (1 year, 6 months).

*Case 127.*—Age 52. Lump noticed in right breast for three months. On examination there was a hard lump, 4 cm. in diameter, just above the nipple. Not attached to the skin. Soft gland palpable in axilla. Radium treatment given on March 17, 1930. On Aug. 27 the lump had disappeared and no glands could be felt. The patient has remained well since (1 year, 1 month).

**2. Intermediate Growths.**—These are still more difficult to define, though they are found in a considerable proportion of the patients treated. In general, the growths are of fair size, though not associated with ulceration, marked contraction, or large secondary glands. In such cases the tumour is likely to be perceptibly smaller after four or five weeks, but the full effect may not be obtained until three months, or more, have elapsed. Growths of this type often have a considerable amount of fibrous stroma which accounts for a good proportion of their bulk. It is remarkable how, in many instances, this fibrous stroma disappears (presumably after the carcinoma cells), leaving the breast in the end quite soft and normal to palpation. As already mentioned, radium treatment does not often result in *fibrosis*, when the dosage has been

correctly gauged. Fibrous tissue actually tends to disappear, so that sometimes a breast which was previously elevated drops to its normal level, and nipples which were retracted become again prominent (*see Cases 15, 59, 68, 73*).

*Case 40.*—Age 57. Lump noticed in left breast for two months. On examination there was a large swelling in the upper part of the breast, adherent to the chest wall and to the skin,  $6 \times 3.5$  cm. in diameter. No enlarged glands detected. Radium treatment on June 8, 1928. Section showed a highly anaplastic carcinoma. By Sept. 5 the tumour had completely disappeared. A small ulcer developed subsequently, but healing took place, and the patient has remained quite well to the present time (3 years, 3 months).

*Case 19.*—Age 70. Lump noticed beneath left nipple for five years. On examination the nipple was retracted and the skin on either side depressed and reddened. Under the nipple was a lump 2.5 cm. in diameter. There was a secondary growth in the axilla, adherent to the skin, 2 cm. in diameter. Radium treatment on Aug. 2, 1927. Both primary and secondary growths had disappeared by Oct. 10, and the patient has remained well since (4 years, 1 month).

*Case 113.*—Age 40. Lump noticed in left breast for four months. On examination there was a swelling,  $5 \times 4$  cm., above the nipple, which was retracted and inverted. No palpable glands. Radium treatment on March 20, 1930. By July 2 the lump had quite gone, and the patient has remained well since (1 year, 5 months).

*Case 130.*—Age 61. Lump noticed in right breast for fifteen months, and another in right axilla four months. On examination there was a hard lump in upper outer quadrant, 3 cm. in diameter, not attached to skin or fascia. Hard gland palpable in right axilla. Radium treatment on Aug. 28, 1930. On Dec. 12 the primary tumour had completely gone. The gland was still palpable. On Feb. 3, 1931, there was no discoverable sign of disease, and the patient is apparently quite well at the present time (1 year).

*Case 15.*—Age 40. Lump noticed in left breast for one year. On examination the breast was elevated and the nipple retracted, with a lump,  $2.5 \times 3$  cm., beneath areola. Tumour attached to the skin and the nipple. Gland palpable in left axilla.

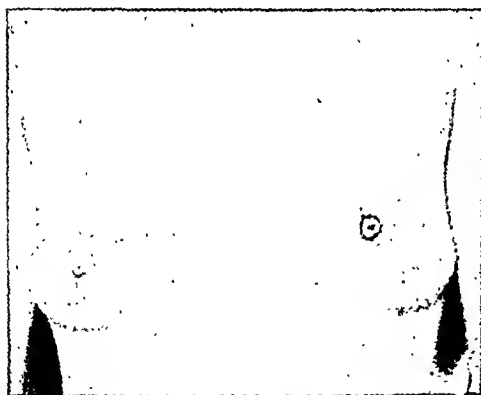


FIG. 316.—Case 15. Before treatment.



FIG. 317.—Case 15. Three years after treatment.

Radium treatment on Feb. 17, 1927. Section showed spheroidal-celled carcinoma with much fibrous tissue. Some puckering of the skin followed with complete disappearance of the tumour and the gland by June 13. The patient has remained well since (4 years, 6 months). (*Figs. 316-318.*)

*Case 62.*—Age 58. Lump noticed in right breast for six months. On examination hard swelling,  $2.5 \times 2$  cm., in upper inner quadrant, attached to the skin. Slight retraction of nipple. Small gland palpable in right axilla. Radium treatment on May 3, 1929. On July 16 most of the tumour had disappeared, leaving a small knob, 1.5 cm., attached to the skin. Gland no longer palpable. All trace of the disease had gone by July 8, 1930 and the patient has remained quite well since (2 years, 4 months).



FIG. 318.—*Case 15.* Position of radium needles.

In all these examples a satisfactory result has been obtained with growths of considerable extent, and it is with the gradual multiplication of cases such as these that real confidence in the treatment begins to grow. These patients have been relieved of all signs of disease and are virtually normal women, without mutilation or serious disfigurement.

**3. Advanced Growths.**—These may be further classified as massive, fungating, ulcerated, or contracting. As is well known, some of the advanced but contracting growths are favourable as regards end-results, because the contraction may denote active and successful resistance by the patient's tissues. On the other hand, they are often not amenable to operation owing to their being adherent to the chest wall, so that they would be universally recognized as suitable for treatment by irradiation. Such growths often



FIG. 319.—*Case 3.* Before treatment.



FIG. 320.—*Case 3.* Two months after treatment.

respond extremely well to radium, and, although too much tissue may have been destroyed by the growth for the breast to be restored to normal, sometimes no trace remains except a permanent depression in the skin. The effect will usually be obtained within five months.

*Case 3.*—Age 43. Lump in right breast noticed for two years. On examination the right nipple was retracted and elevated. There was a very hard growth in the inner upper quadrant,  $5 \times 2.5$  cm. in size. Skin infiltrated, forming a bluish depression, with raised red edges. Growth fixed to pectoral fascia. Enlarged



FIG. 321.—Case 3. Position of radium needles.

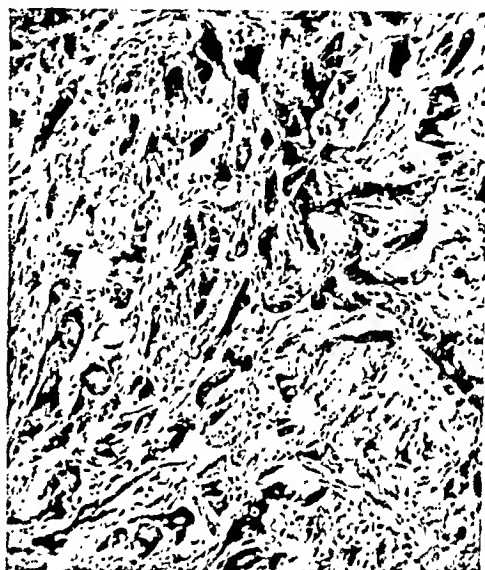


FIG. 322.—Case 3. Section showing fibrous tissue infiltrated with spheroidal cells.

glands felt in right axilla. Radium treatment on May 5, 1925, 27.5 mgrm. being applied to the breast for 8 days. The lymphatic areas were treated in stages. Section removed on May 13 showed much fibrous tissue infiltrated with spheroidal-celled carcinoma cells. On Aug. 10, three months after treatment, no tumour could be detected. The glands disappeared, and the patient is perfectly well at the present time (6 years, 4 months). (*Figs. 319-322.*)

*Case 8.*—Age 57. The only male patient in the series. Irritation and pain in the left breast for one year. On examination there was a lump beneath the left nipple, 2.5 cm. in diameter, attached to the skin and pectoral fascia. Nipple retracted, areola drawn in, and much reduced in size (*Fig. 323*). Skin infiltrated on the mesial side. Palpable gland in axilla. Radium treatment in two stages, on March 23 and April 6, 1926. Section removed on March 23 showed spheroidal-celled carcinoma. The tumour had completely disappeared by Aug. 9, and the patient remained free from recurrence until he died of intercurrent disease on Oct. 16, 1927 (1 year, 7 months).

*Case 11.*—Age 49. Lump noticed in left breast for eighteen months. On examination, in the lower half of the left breast was a large hard indefinite mass, firmly fixed to the chest wall. Skin over it attached and puckered. No glands detected. Radium treatment on Aug. 6, the needles being applied in three layers. Section removed on Aug. 12 showed spheroidal-celled carcinoma, with much fibrous tissue. On Jan. 3, 1927, no tumour could be felt, and the patient has remained free from local recurrence since that date (5 years, 1 month).

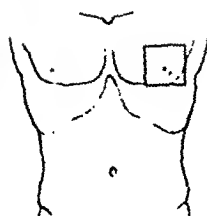
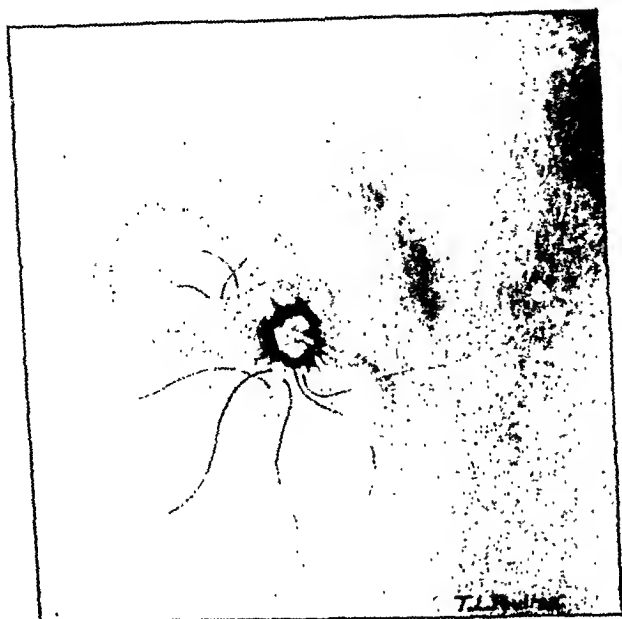


FIG. 323.—Case 8. Carcinoma of male breast before treatment.

*Case 24.*—Age 48. Pucker noticed in left breast for two years. On examination a hard swelling,  $5 \times 2.5$  cm., in inner half of breast, fixed to chest wall. Breast elevated, nipple retracted. Hard gland palpable in left axilla. Radium treatment on Nov. 4, 1927. Section showed spheroidal-celled carcinoma. By Feb. 13, 1928, no tumour could be detected, and the gland had disappeared. No recurrence of the disease since (3 years, 10 months). (Figs. 324–327.)



FIG. 324.—Case 24. Before treatment.



FIG. 325.—Case 24. Four months after treatment.

*Case 27.*—Similar to the above, but a second radium treatment was required for a recurrence near the axilla (see p. 451).

*Case 36.*—Age 68. Lump noticed in left breast for one year. On examination the breast was elevated and the nipple retracted. Above the nipple was a purplish

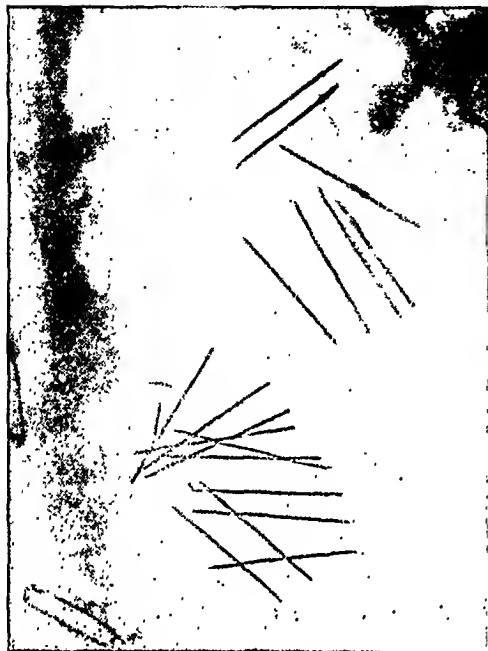


FIG. 326.—Case 24. Position of radium needles.

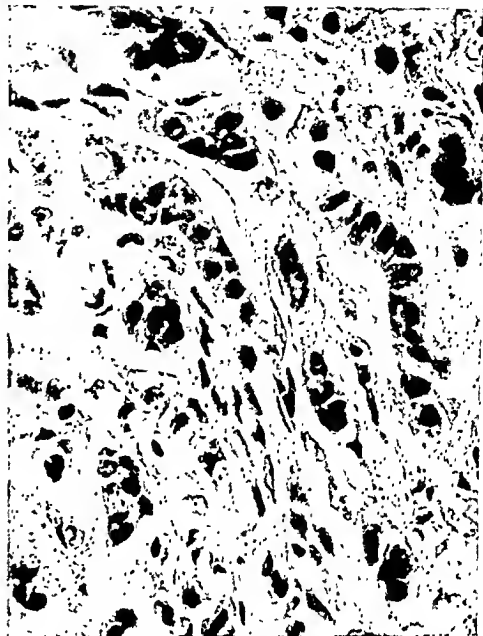


FIG. 327.—Case 24. Section showing spheroidal-celled carcinoma.

lump, 4 cm. in diameter, ulcerated in the centre, edges irregular. Growth about 2.5 cm. thick, fixed to chest wall. No enlarged glands palpable. Radium treatment on May 11, 1928. Section showed spheroidal-celled carcinoma. By Nov. 14 only a small crater remained at the site of the growth with some hard nodules round the margin. On April 11, 1930, the crater was still not quite healed, and a second radium treatment was given. Healing took place soon after. Some hardness remains at the original site, but no evidence of active disease (3 years, 4 months.)

*Case 43.*—Age 63. Lump noticed in right breast for three years, ulcerated nine months. On examination, in the upper outer quadrant was a flat ulcer with a hard raised rim, 5 cm. in diameter. Not adherent. No glands detected. Radium treatment on July 6, 1928. Section showed spheroidal-celled carcinoma. By March 13, 1929, the ulcer was healed and no evidence of disease could be found. The patient died on Dec. 11, 1930, from pulmonary metastases without any sign of local recurrence (2 years, 5 months).

The other categories of advanced growths, massive, fungating, and ulcerated, have been all too common among the patients treated. In fact no fewer than 23 of them have been unscientifically, though expressively, catalogued as 'hopeless' when first seen—that is to say, it was almost inconceivable that they could be benefited by treatment of any kind. Indeed, throughout this investigation no patient has ever been refused treatment except on the ground of obvious visceral or skeletal metastases, so that statistics cannot be regarded as having been vitiated by 'selection'. Nevertheless, the treatment even of apparently hopeless cases has occasionally been rewarded by a remarkably good result, as is illustrated by the following example:—

## RADIUM TREATMENT OF BREAST CANCER 433

*Case 10.*—Age 63. Lump noticed in right breast for three years. On examination, above the nipple was a large purplish mass, 8 × 5 cm. in size. In the centre were two dark-blue soft swellings, and around them the growth was hard and the edges red and raised. Skin infiltrated over the whole area. Growth fixed to pectoral fascia. The axilla was almost completely filled by a hard mass, and some small lumps were palpable in the infraclavicular region. Radium treatment given



FIG. 328.—*Case 10.* At time of first treatment.



FIG. 329.—*Case 10.* Nine months after first treatment.

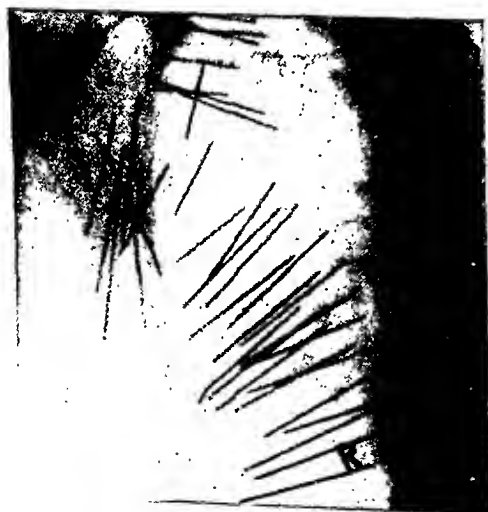


FIG. 330.—*Case 10.* Position of radium needles in first treatment.



FIG. 331.—*Case 10.* Three years after first treatment.

on July 13, 1926, needles being placed deep to the growth to avoid causing necrosis. Section showed spheroidal-celled carcinoma without much connective tissue. The mass continued to shrink for eight months after treatment, and a second treatment was given on May 3, 1927. There was further improvement during the next seven months. Third treatment on Jan. 17, 1928. By March 12 there was no evidence of active disease. A fourth treatment was given on June 7, 1929, for a small recurrent



nodule in the skin, outside the area treated. By Oct. 2 this had disappeared. On July 8, 1930, there was some evidence of reerudescence of growth under the areola, and a surface application of radium was given on Oct. 9. By this date there was a palpable mass in the abdomen, probably an omental metastasis, but the disease has not progressed locally, and the patient feels well (5 years, 2 months). (*Figs. 328-331.*)

Even when the growth has been of massive size it has sometimes disappeared with remarkable rapidity—that is, within three months of treatment; but in other instances a lump of doubtful nature has still remained after five or six months, and it is these that have called for further radium treatment or for local removal. (The evidence derived from these cases is described in detail elsewhere—*see p. 439.*)

Fungating growths, which are sometimes also ulcerated, respond well to treatment, and sometimes the ulcer heals within a few weeks, and the growth completely disappears within four or five months. This type of growth, being conspicuous on the surface, lends itself well to illustration, and provides the most easily demonstrable result. Healing of an obvious ulcer has occurred in six patients, including the first patient treated, who has already been described in detail. Occasionally a small crater, surrounded by some remnant of the growth, has persisted and has called for a local excision, but this may be regarded as a satisfactory result in an apparently unfavourable type.

*Case 41.*—Age 68. Lump noticed in right breast for two years. On examination there was a large ulcerated tumour in the inner upper quadrant, fungating some distance above surface of skin, 6 cm. in diameter. Nipple almost destroyed. Large hard gland in axilla. Radium treatment on June 22, 1928. By Sept. 5 the mass had almost disappeared, but a small ulcer remained in the centre. This was healed by March 5, 1929, but some small nodules remained in the skin. The area of the scar was excised, but the patient died of cerebral hæmorrhage on March 14. Section showed carcinoma cells present beneath the skin.

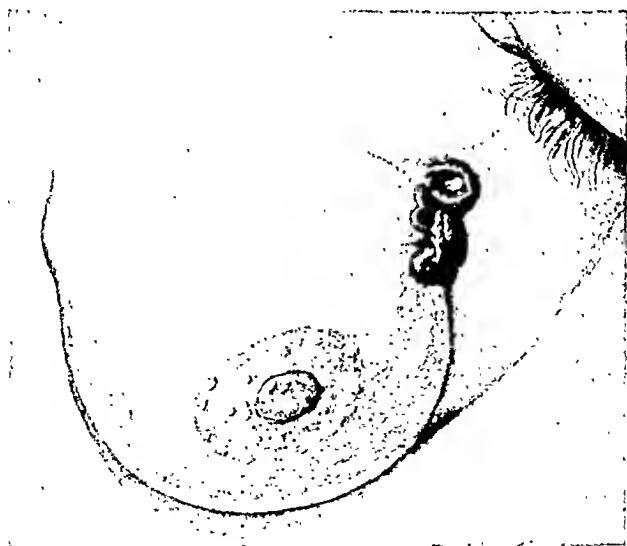


FIG. 332.—*Case 45.* Appearance of tumour before treatment.

## RADIUM TREATMENT OF BREAST CANCER 435

*Case 45.*—Age 40. Lump noticed in left breast for two years. Ulcerated for three months. On examination there was an ulcer,  $4 \times 5.5$  cm., with raised everted edges, and underneath a hard lump,  $5 \times 6.5$  cm., attached to underlying structures. Large gland palpable in left axilla. Inoperable (*Fig. 332*). Radium treatment on

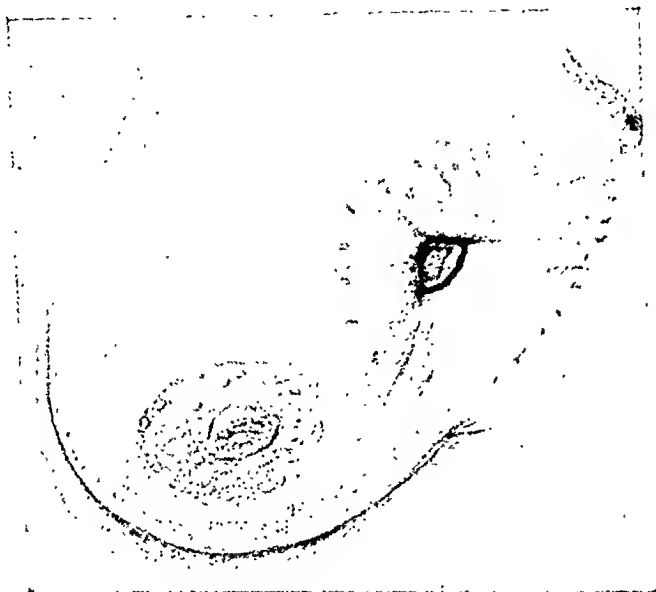


FIG. 333.—*Case 45.* Five months after treatment.

Aug. 23, 1928. The ulcer gradually healed, and four months after treatment the lump in the breast and the axillary gland had disappeared. Five months after treatment the appearance was as in *Fig. 333*. Local condition remained satisfactory, but patient died with spinal metastases on Dec. 16, 1930 (1 year, 8 months).



FIG. 334.—*Case 104.* Before treatment.

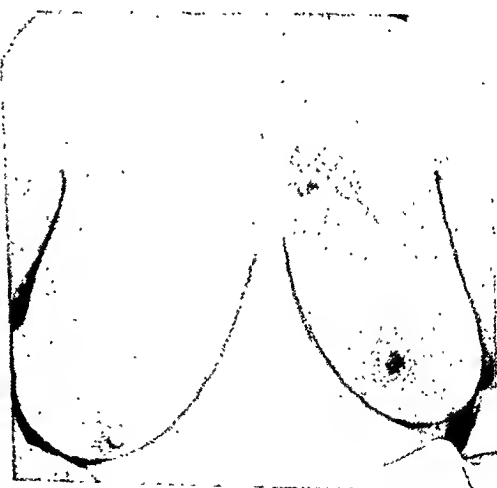


FIG. 335.—*Case 104.* Sixteen months after treatment.

*Case 104.*—Age 55. Lump noticed in left breast for five years. On examination there was an enormous mass, 13 cm. in diameter, in the upper part of the breast,

fixed to deep fascia and to skin, which was partly ulcerated. Large gland palpable in right axilla. Radium treatment on Feb. 27, 1930. Increase of ulceration followed, but by Aug. 27 it was quite healed, and only a remnant of the tumour remained. A second radium treatment was given on this date. On June 24, 1931, nodules seemed to be developing in the scar, and this residual mass was excised on July 2. The wound has healed well and the patient is without signs of disease at the present time (1 year, 6 months). (*Figs. 334, 335.*)

*Case 128.*—Age 73. Lump noticed in left breast for two and a half years. On examination there was a large mass in the upper part of the breast, 7 cm. in diameter, raised above the surface of the skin and ulcerated. Skin infiltrated. No enlarged glands felt. Radium treatment on July 17, 1930, and re-needled on Aug. 11. By Dec. 17 the lump was reduced to 3 cm. in diameter, and was excised on Jan. 8, 1931. The wound healed slowly, but the patient is without signs of disease at the present time (1 year, 1 month).

*Case 149.*—Age 64. Lump noticed in left breast for two years, ulcerated six months. On examination there was a large foul ulcerating mass in the upper half of the breast, 8 cm. in diameter. No glands palpable. Radium treatment on



FIG. 336.—*Case 149.* At time of first treatment.



FIG. 337.—*Case 149.* Two months after first treatment.

Jan. 19, 1931. The ulcer rapidly decreased and was nearly healed by March 23, 1931. Second treatment given on June 6. Further local improvement, but the disease increased behind the clavicle with paralysis and œdema of the arm. (*Figs. 336, 337.*)

*Sloughing* in an advanced growth has been a rare event. It has occurred only three times. In one patient the growth had already invaded a costal cartilage and was ulcerated on the surface, so that sloughing was not unexpected. Although the resulting ulcer never quite healed, the patient remained for long periods free from any sign of active disease.

*Case 12.*—Age 47. Lump noticed in left breast for twelve years. On examination the left breast was found to be pulled up much higher than the right by a contracting growth in the upper inner quadrant, about 5 cm. in diameter, ulcerated in the centre. The areola was contracted, and there was a nodule in the skin below the nipple. A deep furrow in the skin led outward from the growth to a hard lump in the axilla. Radium treatment on Nov. 19, 1926. Section showed spheroidal-celled carcinoma. On Jan. 13, 1927, the whole area of growth was occupied by a shallow ulcer with a sloughing base. The lump in the axilla had disappeared. The ulceration slowly deepened and was ultimately followed by sloughing of the underlying costal cartilage. On July 20, 1928, a second treatment was given for a nodule

## RADIUM TREATMENT OF BREAST CANCER 437

in the skin in the axillary line. This disappeared. On Oct. 25, 1929, a third treatment was given for an enlarged gland in the opposite axilla. On Sept. 3, 1930, cutaneous nodules were appearing on the back, and the patient died on May 25, 1931, with pulmonary metastases (4 years, 6 months).

In another an enormous growth was found. on examination of a section. to be already undergoing spontaneous necrosis. Here, again, it was not unnatural that massive necrosis followed, with eventual healing; the patient remained thereafter free from gross local disease, though she eventually succumbed to skeletal metastases.

*Case 39.*—Age 39. Lump noticed in left breast for one year. On examination the breast was occupied by an enormous tumour, adherent to the pectoral muscle and to the skin. No enlarged glands detected. Radium treatment on June 8, 1928. Section showed the greater part of the growth to be necrotic, though a few carcinoma cells could be recognized. Sloughing of the whole mass followed the treatment. Healing followed within two months. Second radium treatment given for nodules in the skin on April 19, 1929. Patient developed pulmonary metastases and died in January, 1930 (1 year, 7 months).

In the third patient the radium treatment had been preceded by extensive X-ray treatment (*Case 29*). In the fourth patient the tumour was a flat scirrhus growth.

*Case 126.*—Age 57. Lump noticed in left breast for one year. On examination the breast was occupied by a hard irregular mass,  $5 \times 4$  cm., fixed to pectoral muscle and to skin, which was red and crusted. No glands palpable. Radium treatment on July 11, 1930. Sloughing of the tumour followed the treatment, and healing was slow. It was almost healed on May 5, 1931, and there are no signs of disease at the present time (1 year, 2 months.)

### EFFECT OF IRRADIATION ON GLANDS.

In applying radium treatment to carcinoma in other parts of the body, notably in the tongue and mouth, the effect on the lymphatic glands has been found to be unsatisfactory, and but little progress has been made in this part of the problem during recent years. In treating carcinoma of the breast, on the other hand, I have constantly been surprised at the excellent way in which secondary glands have responded to treatment, and the infrequency with which recurrence in glands has taken place, whether they were previously palpable or not. Since the first few patients were treated, the routine irradiation of the lymphatic areas, as detailed above, has never been omitted. The technique, as I have described it, is not as a rule either difficult or dangerous, though it has naturally met with some criticism. The late G. E. Birkett,<sup>7</sup> for instance, believed that it is impossible to irradiate the axilla accurately by implanting needles through the skin, and he held that it should always be done through a wide incision. With this view, after comparatively long experience, I am in complete disagreement as regards the ordinary patient. If there are palpable glands in the axilla, it is quite easy to surround these accurately with the long needles, and the glands will then usually disappear completely and permanently. If there are no palpable glands, it is not difficult to place needles on the four walls of the axilla in such a way that all the lymph glands will be irradiated, and nothing is to be gained

by open incision. I have never yet injured a nerve or vessel in the axilla. I am willing to admit, however, that there is one type of patient—namely, the very stout woman—in whom the procedure does become difficult, and I would even go so far as to say that for the extreme case of this type, radium treatment is altogether unsuitable, though probably this does not apply to more than 2 per cent of the patients presenting themselves.

Recurrence of disease in axillary glands has been noted in only nine patients, and this, considering the advanced stage of the disease in so many of them, is a remarkable observation. Some surgeons of experience even before the advent of radium treatment were beginning to doubt the wisdom of extensive dissection of the axilla, and the results obtained with radium—the most conservative means of treatment available—tend to confirm this view.

Of the nine patients who did have recurrent disease in the axilla several received further treatment. Three are at present free from signs of disease, and the other six are among those who have died.

As regards the supraclavicular glands, here again it appears to me that accurate needling of the glands is not difficult and is effective. G. E. Birkett appeared to regard the introduction of two needles above the clavicle and two below it as theoretically inadequate. The fact remains, however, that I have noted supraclavicular recurrence in only three patients: one of these after further treatment has no palpable glands anywhere (*Case 71*, p. 452); the other two were in an advanced stage of the disease and have died.

Needles have been introduced into the upper three intercostal spaces chiefly on theoretical grounds. I have never seen recurrence take place in this position, or in the area between the sternum and the breast. Neither have I yet seen recurrence take place in the epigastrie region, which I have not been in the habit of treating.

### HISTOLOGICAL EVIDENCE OF THE EFFECT OF RADIUM.

As already mentioned, during the earlier stages of this investigation a specimen was removed from the primary growth in order to prove the nature of the disease. This was done in fifty consecutive cases, the specimen being taken when the needles were removed—that is, after the carcinoma had been subjected to irradiation for a period of at least seven days. The histological appearance of the growth was not obviously altered in the specimens thus obtained, even though the growth was destined, in due course, to disappear. Other evidence, however, has been available from specimens removed at much longer intervals after irradiation.

In six patients it was found after the lapse of several months that small hard nodules still remained in the skin or round the rim of a small residual ulcer. It was obvious in these cases that some carcinoma still remained, and a small area was excised locally. In all of these the presence of carcinoma was verified, though in most of them a large or even inoperable tumour had been reduced to small dimensions. Of the six patients, four are without signs of disease, one has no local recurrence but has metastases in bones, and one has died of cerebral hæmorrhage. The history of one of these patients is peculiar:—

## RADIUM TREATMENT OF BREAST CANCER 439

*Case 21.*—Age 61. Lump under left nipple noticed for at least three years. On examination the nipple had disappeared, leaving an ulcerated cleft. Beneath this was a hard mass,  $2 \times 3$  cm. No enlarged glands detected. Operable. Radium treatment on Sept. 20, 1927. By Jan. 31, 1928, the ulcer was almost healed, but a small rim of hard nodules remained around it. The lump beneath had practically gone. On this date the ulcer was locally excised. Section showed, as expected, carcinoma cells immediately surrounding the ulcer. None were discovered deep. Patient has remained locally cured, though a small ulcer of the skin was excised from the axilla on Dec. 5, 1930, which showed localized carcinoma cells, while a lymph gland in close proximity was free. Patient now apparently well (3 years, 11 months).

This specimen showed disappearance of all the deeper portions of the growth after a single radium treatment, and a small local operation has apparently eradicated the disease in the breast. The recurrence in the skin of the axilla can only be explained by supposing that carcinoma was present in the axillary glands at the time of the first treatment, and that a cancer cell was transplanted into the skin. This is the only occasion on which such an event has been noted.

In eighteen other patients a firm mass has remained in the breast even after two treatments. Owing to the uncertainty as to the nature of these masses the breast has been removed, or a portion of it excised, and this material has furnished important evidence as to the effect of the treatment.

*Case 9.*—Age 60. Lump noticed in left breast for six months. On examination there was a firm lump, 2 cm. in diameter, freely movable. No other signs of disease. The diagnosis was doubtful and the lump was excised locally. It was found to be a localized mass of carcinoma, and radium treatment was given immediately after the operation on May 18, 1926. Eleven months later the growth recurred, and a second treatment was given on April 28, 1927. The lump did not completely disappear, and it was excised on Sept. 23, 1927. A section through the centre of the mass showed fibrous tissue only, and no trace of carcinoma could be found (*Fig. 338*). No local recurrence took place, but the patient died on Oct. 1, 1930, with intrathoracic metastases (4 years, 5 months).

*Case 14.*—Age 56. Lump present in the left breast for at least three years. On examination the upper part of the breast was found to contain a large mass,  $5 \times 4$  cm. and very thick. Skin infiltrated with a shiny reddish mass projecting some distance above the surface. Mass fixed to pectoral fascia. No enlarged glands detected. Inoperable. Radium treatments on Feb. 10 and June 8, 1927. Very great reduction in the size of the mass followed, and the boss on the surface completely disappeared. On Nov. 1 a hard residual lump,  $3 \times 3$  cm., remained, covered by apparently normal skin. Lump removed on this date by a local excision of tissues down to the ribs. Healing by first intention. Section removed on Feb. 16, 1927, showed spheroidal-celled carcinoma with much fibrous tissue. Sections of the residual lump showed that it consisted almost wholly of fibrous connective tissue. On the surface of the mass just under the epidermis—that is, at the farthest point from the radium needles—a thin layer of carcinoma cells remained (*Fig. 339*). Patient remained free from any local recurrence in breast or glands until April 27, 1931, when she died of spinal metastases (4 years, 2 months).

This section appeared to demonstrate conclusively that a very thick mass of growth had been sterilized of carcinoma by two radium treatments, except for a thin layer on the surface, which could have been treated by superficial application. The patient remained locally free from recurrence after excision.

*Case 22.*—Age 61. Lump noticed in right breast for five months. On examination the upper outer part of the breast contained a mass, 3 cm. in diameter, visible on surface, attached to skin. No enlarged glands detected. Operable. Radium treatment on Oct. 25, 1927. Section removed on Nov. 1 showed spheroidal-celled carcinoma. By April 3, 1928, the original mass had completely disappeared. On April 9, 1929, there was apparently a recurrence in the same situation. Second

radium treatment given on June 28. By Nov. 5 the mass was somewhat smaller, but stationary. Some deep X-ray treatment given on Dec. 19. Lump remained almost unaltered, and the breast was locally removed on July 3, 1930.



FIG. 338.—Case 9. Section through area of growth seven months after second treatment. No carcinoma found.

Careful examination of the specimen failed to show any carcinoma cells anywhere. It is possible that the second radium treatment and X-ray treatment were really superfluous. The patient remains quite well to the present time (3 years, 10 months).

*Case 38.*—Age 77. The right breast contained a large mass, 7 cm. in diameter, at least 3 cm. thick. Attached to the skin over a wide area, but not to deep fascia. No enlarged glands detected. Radium treatment on June 1, 1928. Section showed spheroidal-celled carcinoma. On Nov. 19 a lump remained, but only half the size of



FIG. 339.—*Case 14.* Section through residual mass six months after second treatment. Carcinoma cells found subcutaneously only.

the original mass. This was locally excised on Dec. 7, and the patient has remained free from disease since that time (3 years, 3 months).

On careful examination of the specimen removed no trace of carcinoma could be discovered. The residual mass consisted entirely of fibrous tissue (*Fig. 340*).



*Case 42.*—Age 58. Lump noticed in right breast for two years. On examination, found to have a large swelling, 6 cm. in diameter, in upper inner quadrant of breast, fixed to skin and pectoral fascia. Hard enlarged gland, 2 cm. in diameter, in right axilla. Operable. Radium treatment on June 26, 1928. Section showed spheroidal-celled carcinoma. By Jan. 30, 1929, both the lump in the breast and the enlarged gland had completely disappeared. On July 16, 1929, a recurrence was detected

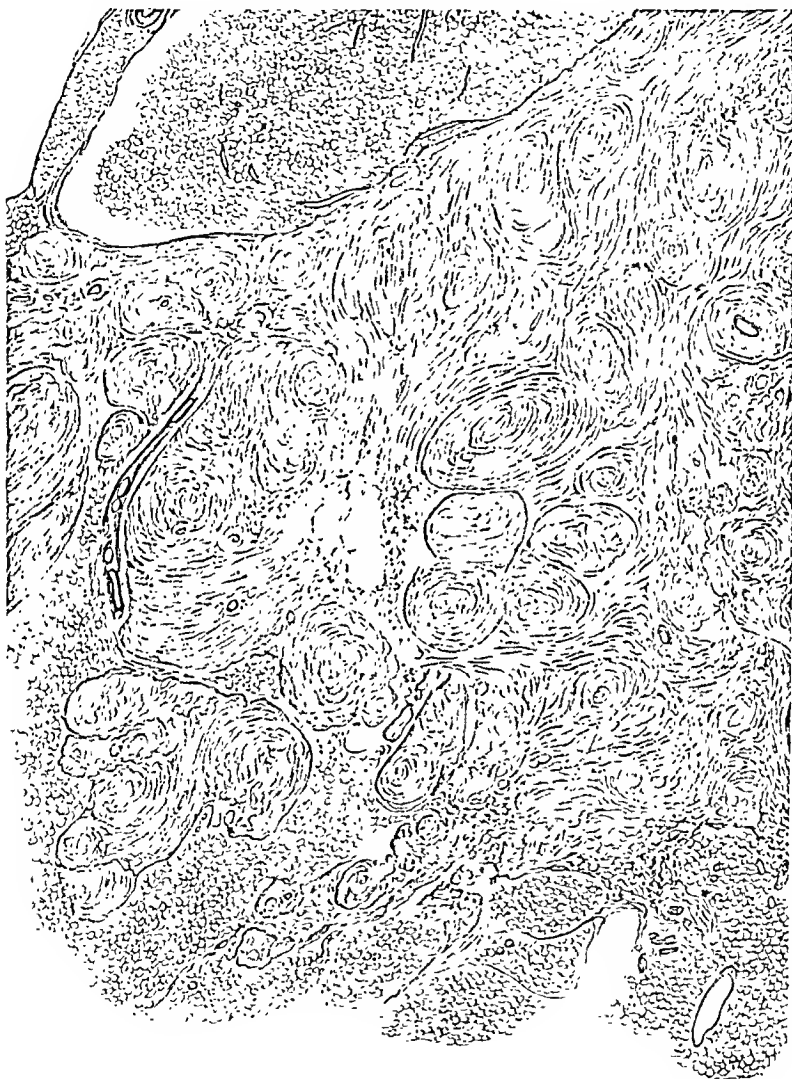


FIG. 340.—*Case 38.* Section through site of massive growth six months after treatment. No carcinoma found.

between the breast and the axilla in an untreated area. Second radium treatment given on Aug. 1. On Nov. 5 patient appeared to be still not free from disease, and the breast was removed locally on Nov. 21. Examination of the specimen showed no sign of carcinoma in the area originally treated. A few isolated carcinoma cells were found at the edge of the specimen. Patient has remained without recurrence since (3 years. 2 months).

## RADIUM TREATMENT OF BREAST CANCER 443

The specimen showed almost complete sterilization of the breast, but in this instance the error had been made of not treating the breast and axilla in continuity, recurrence taking place in the interval between them.

*Case 54.—Age 57.* Lump in right breast noticed for four months. On examination, in outer part of the breast was a tumour, 3 cm. in diameter, attached to nipple. Hard enlarged gland in right axilla. Operable. Radium treatment on March 1, 1929. By July 16 no tumour or glands remained. On May 6, 1930, some evidence of recurrent growth detected. Local removal of breast on July 10. Patient has remained free from disease since (2 years, 6 months).

Examination of the specimen showed a small localized area of carcinoma in the centre of the breast. Otherwise free from disease.

*Case 76.—Age 50.* In the left breast was a large mass, 8 cm. in diameter, occupying the greater part of the gland. Attached to the skin. Nipple retracted. No enlarged glands detected. Radium treatment on July 19, 1929. On Jan. 9, 1930, a small residual lump was still present, and a second radium treatment was given. The lump became somewhat smaller, but was still present on Sept. 29, when the breast was locally removed. The patient is now without signs of disease (2 years, 1 month).

On examination of the breast no trace of carcinoma could be found. The residual lump appeared to be due to a small patch of fibrosis and fat necrosis.

*Case 79.—Age 55.* Lump noticed in right breast for six months. There was a large indefinite tumour, about 6 cm. in diameter, beneath the nipple, which was retracted. Slight patchy discoloration of the skin. No enlarged glands palpable. Operable. Radium treatment on Aug. 28, 1929. By Dec. 10 no tumour was palpable, and the nipple was almost normal. On Feb. 26, 1930, it was noticed that one of the small patches on the skin was slightly thicker than before. The breast was

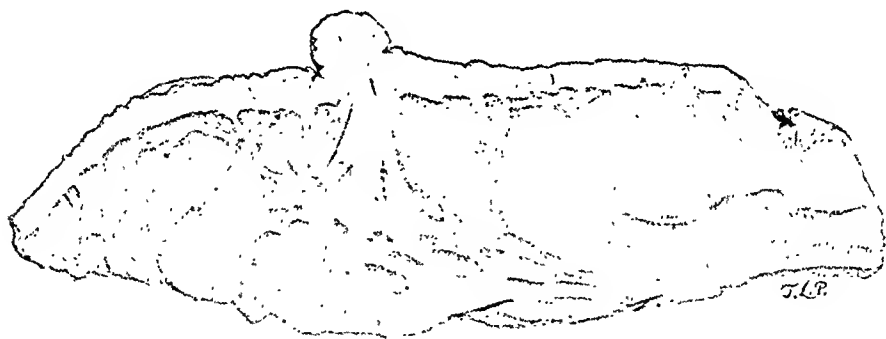


FIG. 341.—*Case 79.* Slice through breast eight months after treatment for a massive carcinoma.

removed locally on May 1. In September, a precautionary surface application was given to the right side of the chest. The patient now has signs of thoracic metastases (September, 1931).

The examination of the specimen gave interesting results. The large mass previously present had completely disappeared, so that a slice through the breast looked quite normal (*Fig. 341*). No trace of carcinoma could be found anywhere in the breast. A section through the small patch in the skin showed, however, carcinoma cells present in the dermis in this area. Presumably, therefore, the disease could have been completely eradicated by a superficial application after the interstitial irradiation.

*Case 82.—Age 42.* Lump noticed in left breast for a few days. On examination the left nipple was found to be slightly elevated and retracted. There was a large hard lump in the upper part of the breast, about 7 cm. in diameter, attached to the

nipple, but not to the skin or deep fascia. Operable. Radium treatment on Sept. 10, 1929. By Nov. 5 the breast was quite soft and no tumour remained. On Oct. 1, 1930, it was noticed that there was some thickening to be felt on the deep surface of the breast in its upper part. The breast was removed locally on Oct. 30. The patient is at present without signs of disease (2 years).

Examination of the specimen showed no sign of carcinoma anywhere, the thickening being due to fibrosis only.

*Case 106.*—Age 49. Lump noticed in right breast for six months. There was an elongated hard mass extending from one side of the breast to the other, at least 8 cm. long. Nipple retracted, and skin dimpled on outer side. No enlarged glands detected. Operable. Radium treatment on March 7, 1930. By Sept. 10 the breast was quite soft, though there remained some soreness of the nipple. This afterwards increased, and there appeared to be a small lump beneath the nipple. The breast was removed locally on Nov. 25. The patient is free from disease at the present time (1 year 6 months).

Examination of the specimen showed active carcinoma cells immediately beneath the nipple.

*Case 111.*—Age 50. Both breasts very large. Lump noticed in upper part of right breast for one month. On examination the right nipple was found slightly elevated, but not retracted. In the upper outer quadrant was a very large mass, about  $7 \times 5$  cm. in diameter, and very thick. Two hard glands felt in right axilla. Operable. Radium treatment on April 11, 1930. Left breast removed at same time owing to its weight. By July 8, no tumour could be felt and no glands were palpable. Right breast removed on Feb. 6, 1931, although no recurrence detected, owing to the large size both of the breast itself and of the original tumour. In the site of the growth on the deep surface of the breast was found a small carcinomatous area, 1.5 cm. in diameter. No other evidence of disease found. A very large growth had, therefore, been almost, but not quite, eradicated by a single treatment. The patient is quite well at the present time (1 year, 5 months).

The evidence obtained from these eighteen patients is on the whole a satisfactory testimony to the powers of radium. Admittedly, the necessity for removal argues that radium is not all-powerful or that the present technique is at fault. Infallibility is not claimed, however, for the method, and the fact remains that in 7 of the 18 specimens no residual carcinoma was found. In 9 of the remaining 11 a large carcinomatous mass had been reduced to small dimensions. In only 2 instances had the disease spread widely after irradiation. It is of interest to notice that, although nearly all the patients were in an advanced stage of the disease and would be looked upon unfavourably as regards prognosis, they do not appear to have lost by the method of treatment that was used. Of the 18 patients, 17 are alive, and 14 of them are without any signs of disease at the present time—that is, for periods up to 3 years, 9 months. One patient died of the disease, one has skeletal metastases, one thoracic metastases, and one metastases in the skin. Removal of the breast after irradiation appears therefore to be a legitimate method of combining radium treatment with surgery.

### CLINICAL EVIDENCE OF CURE.

In assessing the results of radium treatment of carcinoma of the breast I have consistently tried to avoid using the word 'cure' without qualifications. Sometimes, when all signs of disease have disappeared, I have said that the patient was 'apparently cured', being aware that in this disease recurrences

have been known to appear after the lapse of fifteen or even twenty years after surgical treatment. At other times, so sceptical have I been, that I have stated that there is no such thing as 'cure' in this disease, the patient being liable to develop recurrences for the remainder of her days. Never-

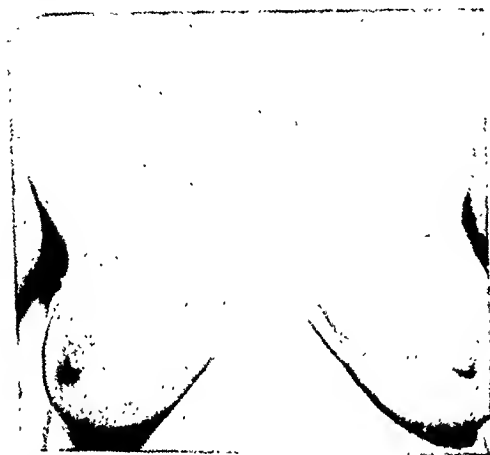


FIG. 342.—Case 23. Before treatment.

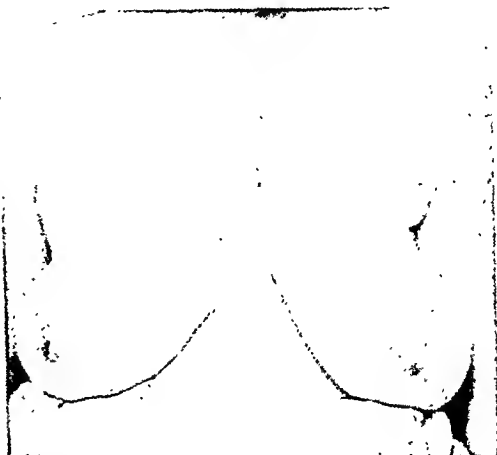


FIG. 343.—Case 23. Three years after treatment.

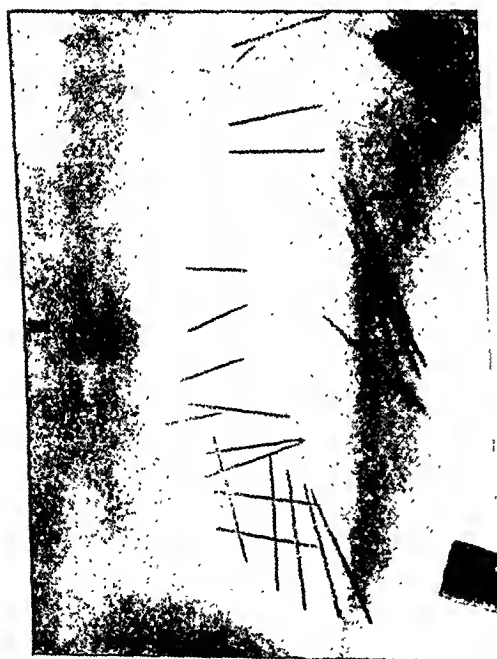


Fig. 344.—Case 23. Position of radium needles.

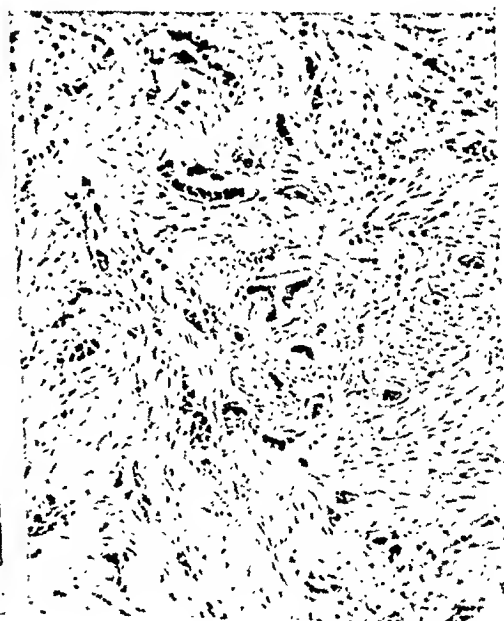


FIG. 345.—Case 23. Section showing fibrous tissue with spheroidal-celled carcinoma.

theless. the histological evidence adduced above, seems to show that 'local cure' can be produced by radium, careful investigation being unable to detect any evidence of carcinoma cells remaining. But no pathological investigation can be minute enough to *prove* that no isolated cells or clumps of cells

remain in the tissue. Some other proof of cure must therefore be sought, and I believe that this may be founded on a physiological basis. The extreme malignancy of carcinoma of the breast when associated with pregnancy or lactation is well known; it is shown by the increased rapidity of growth of the primary tumour and by the unusually quick appearance of metastases in other parts of the body. It is believed that these phenomena are due to local hyperæmia in the breast, and to the profound stimulus applied to the epithelial cells of the mammary gland by the hormone from the corpus luteum in the circulation. If these circumstances were to arise in a woman who had a carcinoma of the breast treated by radium, the stage would be set for the clinical experiment which would apply the severest test possible to the efficacy of the treatment. Actually these circumstances have arisen in the following instance:—



FIG. 346.—Case 23. Three years after treatment.

*Case 23.* Age 44. Small lump noticed in inner part of left breast for two years. On examination, a hard mobile lump, 3 cm. in diameter, was felt in the inner half of the breast, attached to the skin, which was puckered (Fig. 342). Nipple slightly elevated. No enlarged glands detected. Operable. Radium treatment on Oct. 25, 1927. Specimen removed for section showed spheroidal-celled carcinoma with proliferation of epithelium in neighbouring acini. By Feb. 13, 1928, the tumour had disappeared, leaving a small area of thickening. In December the patient became pregnant and was seen five months later. There were no signs of recurrence, and the pregnancy was allowed to proceed to term. The child was fed at both breasts, the left breast lactating normally. There has been no sign of recurrence of the disease, and the patient is quite well at the present time (3 years, 10 months). (Figs. 342-346.)

The history of this patient seems to be as nearly a *proof* that carcinoma of the breast can be cured by a single treatment with radium as can be attained—better evidence even than the passage of twenty-five years after the treatment, for a supposed local recurrence after this interval might really be another independent growth.

Up to the present time this combination of circumstances has not occurred in a second patient. Another woman, age 38, reported to me eighteen months after an apparently quite successful treatment of a small and early carcinoma in the axillary tail of the right breast, with an enlarged gland in the axilla, that she had missed one period. There was no change in the breast, but I felt it my duty in the present state of knowledge to advise that the pregnancy be terminated.

## RADIUM TREATMENT OF BREAST CANCER 447

*Case 89.*—Age 37. Lump noticed in the axillary tail of right breast for a short time. On examination, a small, hard, mobile lump,  $2 \times 1.5$  cm. in diameter, in the axillary tail, not attached to skin or fascia. Fairly large gland palpable in the right axilla and some smaller ones just palpable. Radium treatment on March 1, 1929. By June 12 no trace of the lump remained and the breast felt quite normal. No glands palpable. On Sept. 16, 1930, the patient consulted me because she had found herself to be pregnant. The pregnancy was terminated immediately. The patient has remained quite well since (2 years, 6 months).

### FAILURES.

Radium treatment is a form of local treatment, and failure to achieve local extirpation of the disease is therefore the only kind of failure that need now be considered. The possibility that dissemination may be actually stimulated by radium is discussed elsewhere (p. 458). Failure interpreted in this sense may be of several types, which will be considered separately.

1. The disease may recur actually *within the area treated or in close proximity to it*, and in such cases complete failure to control the disease may have to be admitted. Sometimes, as has also been recorded by other observers, there may be at first such striking improvement that the tumour may disappear completely and the breast become almost normal. Then recurrence takes place, and the fresh growth may progress very rapidly. Complete failure of this kind I have registered in five instances. In one of these the dose given was undoubtedly inadequate, the time having been only four days instead of the usual seven.

*Case 50.*—Age 39. Lump noticed in right breast for one year. On examination a hard irregular mass beneath areola was felt, 5 cm. in diameter and 3 cm. thick. Skin adherent and thickened. Nipple retracted. No palpable glands. Radium treatment on Feb. 5, 1929, and owing to fear of sloughing taking place, the needles were removed at the end of four days. Regression of tumour was rapid, though there was some oedema of the skin remaining on May 7. Two months later the breast was again found to be occupied by a large tumour. A second radium treatment was given on Aug. 5. The tumour again regressed, but the patient showed signs a month later of thoracic and spinal metastases, and she died on April 8, 1930.

In one the breast was extremely fat, a type of patient which I have discussed elsewhere, and the extent of the disease when treatment was begun was greatly underestimated.

*Case 53.*—Age 47. Lump noticed in left breast for one year, and treated by local injections by Dr. Shaw Mackenzie. On examination a hard lump was felt on the outer side of the left breast, of at least 4 cm. diameter, but difficult to estimate owing to the very large size of the breast. The skin was beginning to be dimpled, and a hard gland could be felt in the axilla. Radium treatment given on Feb. 20, 1929. On June 5 the greater part of the lump had disappeared, but some irregularity remained in the upper part of the breast. The axillary gland was smaller. On Sept. 24 the disease was obviously extending, and the breast was removed. Deep X-ray treatment was also given, but the patient died on June 21, 1930 (1 year, 4 months).

In one (*Case 9*) an injudicious local excision was done before treatment, which was otherwise unsatisfactory. In only two cases (*Cases 13 and 32*) was the failure complete.

*Case 32.*—Age 47. Lump noticed in right breast for nine months. On examination a hard lump was felt in the right breast, 4 cm. diameter, attached to skin. Enlarged glands palpable in axilla. Radium treatment given on April 18, 1928, for 9 days. A specimen was removed for section in my absence, through a very deep and long incision, which may have prejudiced the result. On June 5 the lump was smaller, but was increasing again under the incision scar by July 3. A second treatment was given on Jan. 18, 1929, after which the growth did not increase for three months. By May 7 it was again increasing. Deep X-ray treatment was given without benefit, and the patient died with generalized metastases in October, 1930.

*Case 13.*—Age 51. Dimpling of the skin on the outer side of the right breast noticed for eleven months. On examination a hard mass, 8 cm. in diameter, was felt in the outer half of the breast, with retraction of the nipple and adhesion of the skin. Hard gland felt in right axilla. Radium treatment given on Dec. 21, 1926. Section showed spheroidal-celled carcinoma. By March 7, 1927, the tumour and axillary gland had disappeared, though some indefinite thickening remained in the breast. On Sept. 12 two nodules had appeared in the skin on the mesial side of the area previously treated. Second radium treatment given on Sept. 29, followed by disappearance of the nodules. On Jan. 9, 1928, a new lump had appeared between the two areas treated. A local excision of breast showed active carcinoma in the last lump, and some groups of carcinoma cells at the edge of the first area treated. The wound healed slowly and developed a streptococcal infection after the patient had left hospital, and she died on Feb. 18. Post-mortem examination showed no evidence of carcinoma in the chest wall, glands containing carcinoma in the left axilla, above both clavicles, and along the left side of the abdominal aorta.

Much has been written of varying radio-sensitivity in different types of growth in the breast. Such variations certainly do exist, but so seldom is a tumour really insensitive that I have not attempted any 'grading' of the tumours. In the following instance there appeared to be an unusual degree of insensitivity, the breast and the tumour both being small, and therefore easy to irradiate.

*Case 78.*—Age 46. Lump noticed in left breast for six weeks. On examination there was a prominent swelling immediately above the nipple, 6 cm. in diameter, apparently partly cystic. No glands detected. Radium treatment on Jan. 18, 1929. By March 5 the lump had almost disappeared. On Sept. 6 the swelling was found to have reappeared and was almost exactly as before treatment. It was locally excised, and the specimen showed a cyst with many active carcinoma cells in the wall. On April 10, 1931, the patient was found to have a second tumour similar to the first. The remainder of the mammary gland was removed together with the lower axillary lymphatic glands. The specimen showed a carcinomatous cyst exactly like the first. The lymphatic glands were free from disease. The patient is quite well at the present time (2 years, 7 months).

In this instance a carcinoma of a somewhat unusual type appeared to have been almost unaffected by the radium, the second carcinomatous nodule having appeared within the area treated.

2. The disease may recur *outside the area treated*. This form of failure has been registered in nine patients, and the lesson to be learned from it is obvious.

*Case 51.*—Age 43. Small lump noticed in left breast for two months. On examination, slight flattening was found at the lower margin of the breast, which was somewhat pendulous and very large. The nipple was slightly retracted. One

## RADIUM TREATMENT OF BREAST CANCER 449

gland palpable in left axilla. Radium treatment given on Feb. 9, 1929. Treatment was given only to the lower half of the breast, and to the subpectoral region, axilla, etc., a large gap being left in the upper portion of the breast, which appeared to be free from disease. There was evidence of a diffuse extension of the disease in this untreated part of the breast on Sept. 12, 1930, 1 year and 7 months after the initial treatment. Removal of the breast was advised, but the patient refused. A second radium treatment was given on Sept. 19. By Dec. 2 great improvement had taken place, but soon afterwards the disease recurred extensively throughout the breast. The breast and axillary glands were removed on June 19, 1931, and deep X-ray therapy was given to a wide area surrounding. The patient appears to be quite well at the present time (2 years, 7 months).

*Case 75.*—Age 60. Lump noticed in right breast for three weeks. On examination there was a hard lump in the outer half of the breast, 3 cm. in diameter, attached to the skin, which was slightly dimpled. Gland palpable in lower part of axilla, but not hard. Radium treatment given on July 11, 1929. The tumour had disappeared and the breast felt quite normal by Sept. 7. No gland palpable in axilla. On Feb. 16, 1930, some thickening in the untreated part of the breast was noted. This had increased two months later, and a local removal of the breast was done on April 23 instead of giving a second radium treatment.

On examination of the specimen the part of the breast treated was found to be free from disease, but carcinoma was found in the outer area. Following removal of the breast radium was inserted under the whole of the area of operation. There has been no local recurrence, but the patient has signs of thoracic metastases.

*Case 112.*—Age 57. Lump in left breast noticed for one year. On examination a hard mass felt in the inner upper quadrant about  $5 \times 7.5$  cm. Skin dimpled over it. Nipple slightly retracted and deviated. No glands palpable. Patient very stout and breast very large. Radium treatment on April 10, 1930. By Aug. 27 the mass had almost gone. On Oct. 15 there was found to be evidence of extension of the disease near the nipple in an untreated area. A gland could also be felt in the axilla. Second radium treatment on Oct. 23, 64 mgrm. being used for the breast, whereas only 42 mgrm. were used on the first occasion. By March 11, 1931, no sign of local disease could be discovered, but the patient now has pelvic metastases.

These examples illustrate the fact that it is easy to concentrate attention too much upon the main mass of growth, and to forget that secondary dissemination occurs *within* the mammary gland as well as outside it, as already mentioned. Although I have always aimed at treating a considerably larger area of breast than is occupied by the palpable tumour, I have sometimes failed to carry this far enough, and latterly I have practised a wider distribution of the radium than before. Properly the whole breast and the underlying fascia should be treated as a routine, and except in the largest breasts this is being carried out at the present time. It is partly for this reason that I have so much emphasized the importance in technique of using the longest needles and placing them largely beneath the breast. Only in this way can the requisite area of breast and fascial planes be adequately treated. If the breast is not very thick, this distribution will irradiate the tumour as well as the breast. If the breast is a thick one, it may be necessary to insert additional needles more superficially in the neighbourhood of the growth.

Further than this, it is of some importance to remember the main lymph channels which run round the outer border of the pectoralis major muscle on their way to the axillary glands. Examination of the X-ray records of



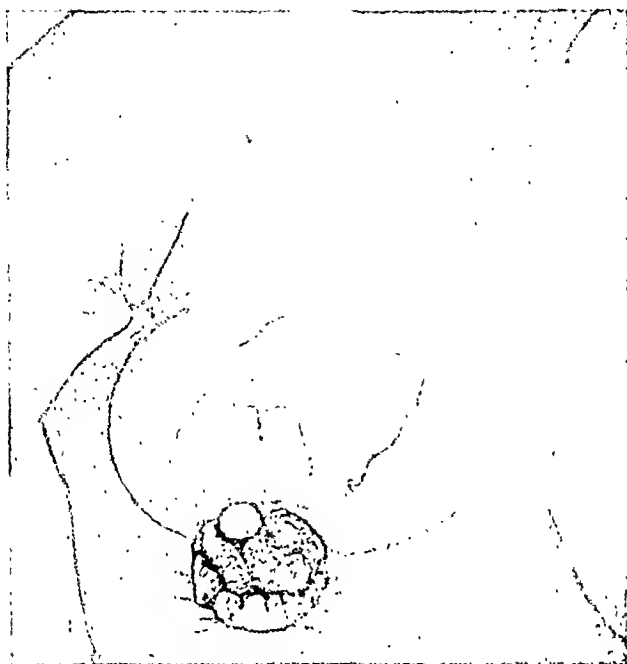


FIG. 347.—Case 27. Appearance of tumour before treatment.

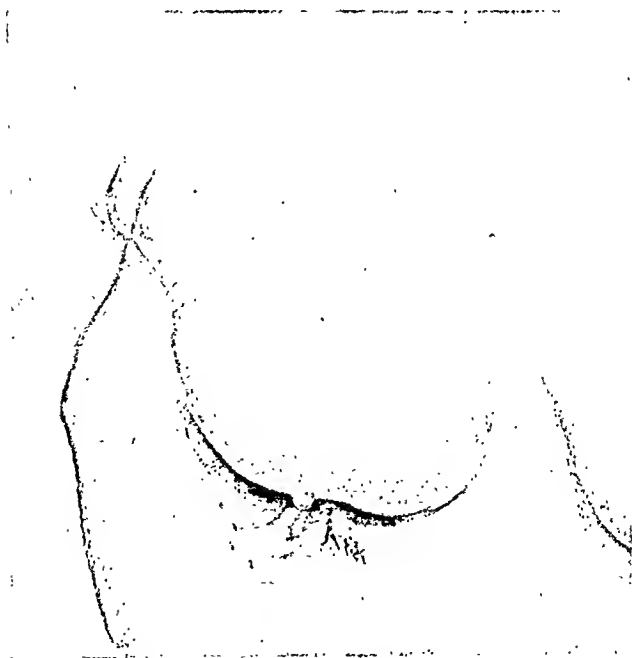
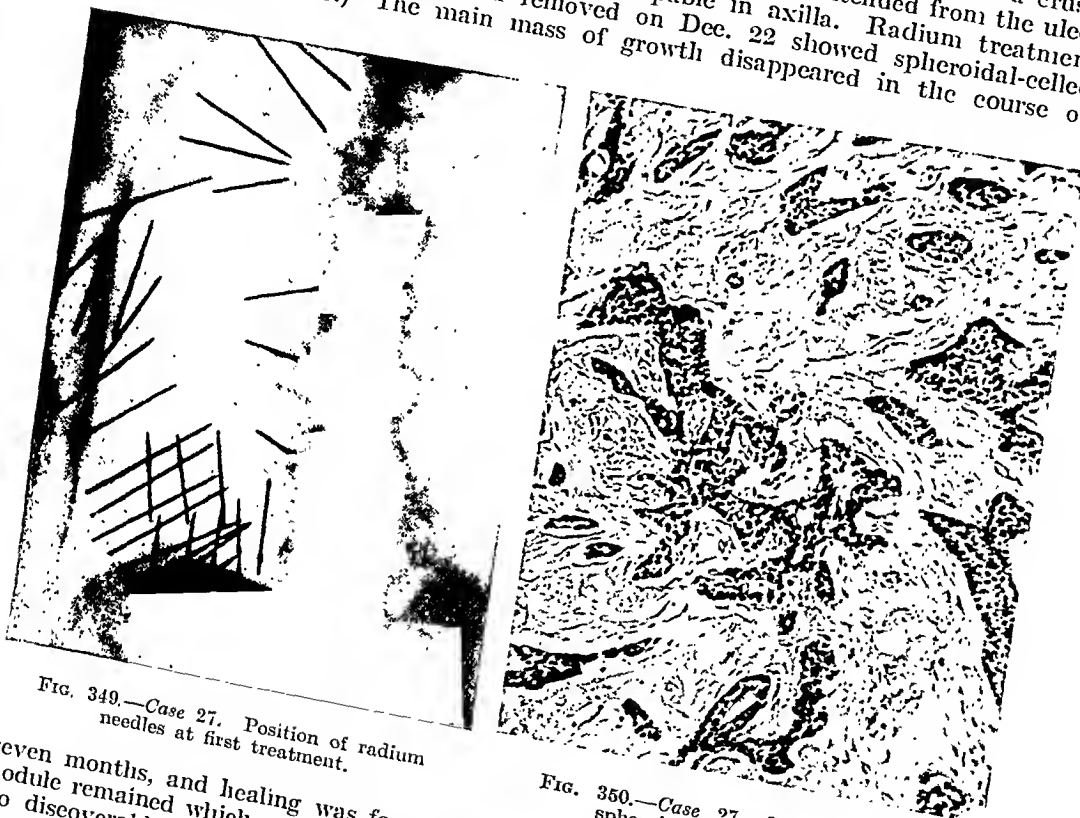


FIG. 348.—Case 27. One year after treatment.

# RADIUM TREATMENT OF BREAST CANCER 451

my earlier patients shows that I have sometimes been guilty of leaving this 'pectoral gap' between the breast and the axilla untreated. Retribution has occasionally followed, for in two patients recurrence has taken place exactly in this untreated gap. The needles should therefore be inserted in unbroken series from the breast to the upper axilla.

*Case 27.*—Age 62. Lump noticed in the right breast for five years. On examination the right breast was elevated and smaller than the left. Nipple retracted. In the inner lower quadrant was a bright-red area, the centre covered with a crust, diameter about 5 cm. (*Fig. 347*). A hard mass,  $3 \times 2$  cm., extended from the ulcer towards the axilla. Several small glands palpable in axilla. Radium treatment given on Dec. 16, 1927. Section removed on Dec. 22 showed spheroidal-celled carcinoma (*Fig. 350*.) The main mass of growth disappeared in the course of



*FIG. 349.*—*Case 27.* Position of radium needles at first treatment.

*FIG. 350.*—*Case 27.* Section showing spheroidal-celled carcinoma.

seven months, and healing was found to be complete by July 18, 1928. A tiny nodule remained which eventually disappeared, and on Jan. 11, 1930, there were no discoverable signs of disease. Needles had been placed under the pectoral muscle (*Fig. 349*), but between these and the needles under the breast a gap was left, and exactly in this position a recurrent nodule was found on Sept. 3. A second radium treatment was given on Oct. 3 to the whole tract from the upper part of the breast to the axilla. On March 3, 1931, the nodule had disappeared, and the patient has remained free from disease since that date (3 years, 8 months).

A second instance of recurrence in the pectoral gap was *Case 42*, already described in detail on p. 442.  
3. A third form of failure is shown by the appearance of numerous *skin nodules within the area treated*. This event has been rare, having been seen only in two patients, but its occasional occurrence is an argument in favour

of Cade's method of supplementing interstitial irradiation by surface treatment.

*Case 34.*—Age 68. A dimpling noticed in the skin of the left breast for two years. On examination the left breast was elevated and the skin drawn in several places. Nipple retracted. The whole of the upper part of the breast was occupied by an irregular mass, about 7 cm. in diameter, attached to the deep fascia. Glands palpable in left axilla. Radium treatment on March 27, 1928. Section removed on May 4, showed spheroidal-celled carcinoma. By Nov. 14 the whole of the original mass had disappeared, but there were nodules in the skin below and to the inner and outer sides. The whole area was treated by a second insertion of needles on Jan. 4, 1929. Two months later all the nodules had disappeared, and there was no local evidence of disease. The patient died on April 4, 1930, of spinal metastases.

*Case 70.*—Age 60. Thickening noticed in right breast for several weeks. On examination the nipple was retracted and the skin puckered below it. A hard diffuse mass occupied the greater part of the breast, about 6 cm. in diameter. No palpable glands. Radium treatment on June 18, 1929. By Sept. 7 no tumour could be detected. A year later a widely spread crop of skin nodules had appeared over the breast, and the patient had also been having signs of pulmonary metastases. A surface application of radium was given to the area affected, but the patient died with evidence of widespread metastases on Nov. 27, 1930.

4. The fourth type of failure is seen in those patients who return, after treatment, with *enlarged glands in the axilla*, either following disappearance of glands previously enlarged, or as a new event. My technique for treating the axilla has met with some criticism, as already mentioned, but the answer to this is the remarkable effect almost always obtained when the glands are already enlarged, and the rarity of recurrence. This event has been noted only in 9 patients, but when it occurs it is of serious import, 6 of these patients having died. The appearance of glands above the clavicle is even rarer. It has been noted in only 3 patients, in 1 of whom the glands have disappeared after further treatment.

*Case 71.*—Age 67. Lump in the left breast noticed for six weeks, in inner lower quadrant, 5 cm. in diameter. Attached to skin, with enlarged glands in the axilla. Radium treatment given on June 21, 1929. Three months later the tumour in the breast had disappeared: one soft gland still palpable in the axilla. On June 5, 1930, one year after treatment, there was no sign of recurrence in the breast, but enlarged glands were present in the axilla and above the clavicle. Further treatment was at first considered useless, but two months later the patient was still without other signs of metastases, and a second radium treatment was given on Sept. 2 to the breast and gland areas. By Nov. 21 all enlarged glands had disappeared, and the patient still seems to be quite well (2 years, 2 months).

### OVERDOSE.

As already suggested, the dosage employed in this form of radium treatment has been arrived at by somewhat empirical methods. It has thus happened that, during the earlier stages of acquiring experience, the dose has occasionally been excessive, either by reason of too great local concentration of the radium, or as the result of ill-judged zeal in dealing with apparent failures. A dose that was too great at the initial treatment is illustrated by the following case:—

# RADIUM TREATMENT OF BREAST CANCER 453

*Case 7.*—Age 57. Lump noticed in left breast for four years. On examination there was a hard tumour filling the breast, with a prominent lump near the areola; skin over this red and glazed. Nodules in the skin in inner part of breast and over the sternum. An enlarged gland palpable in axilla. Radium treatment given on

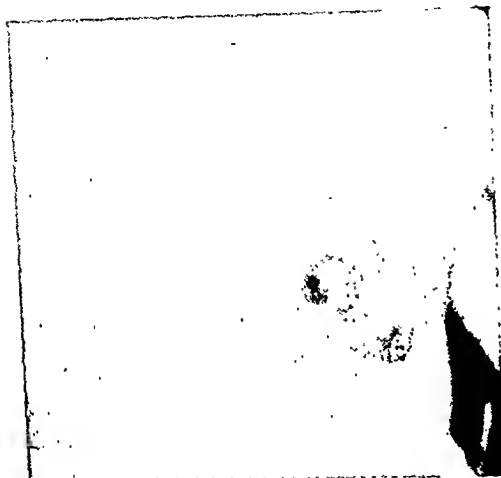


FIG. 351.—*Case 7.* Before treatment.



FIG. 352.—*Case 7.* Radium burn seven weeks after treatment.

Aug. 12, 1925, a total of 79 mgrm. being inserted beneath the breast and its neighbourhood for 7 days. The treatment was followed by a very severe radium burn, with late necrosis of the primary growth. The ulcer never quite healed, and skin nodules afterwards appeared outside the area treated. The patient died nine months after treatment. (*Figs. 351–353.*)

Instances of injudicious second treatments are given by the following:—

*Case 16.*—Age 62. Lump noticed in right breast for three months. On examination there was a very hard lump, 6 cm. in diameter, in the inner upper quadrant, with dimpling of the skin. An enlarged gland palpable in right axilla. Radium treatment on April 14, 1927, a total of 51.5 mgrm. being applied to the breast in two layers. Three months later the tumour seemed to have disappeared, but the breast had undergone a remarkable degree of shrinkage. One year after treatment the patient was thought to have a recurrence in the upper part of the breast. This may have been due only to fibrosis, but a second treatment was given on April 12, 1928, 48 mgrm. being applied to the breast in two layers for 8 days. Further shrinkage took place, and late radium necrosis began two years after the second



FIG. 353.—*Case 7.* Position of radium needles at first treatment.

treatment. The necrosis extended into the axilla, and very deeply into the chest wall. The patient suffered great pain, and died on Jan. 16, 1931, without definite evidence of recurrence of the carcinoma anywhere.

*Case 31.*—Age 58. Lump noticed in right breast for fifteen months. On examination a large spherical tumour, 6 cm. in diameter, was felt in the upper outer

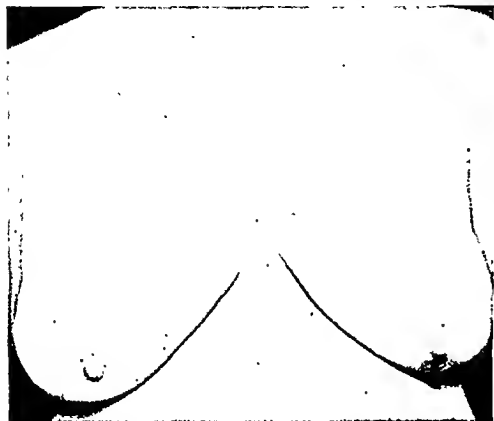


FIG. 354.—*Case 31.* Before treatment.



FIG. 355.—*Case 31.* Sixteen months after first treatment.

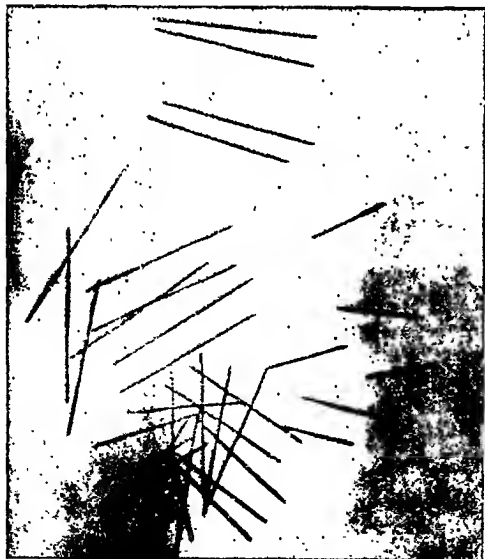


FIG. 356.—*Case 31.* Position of needles at first treatment.

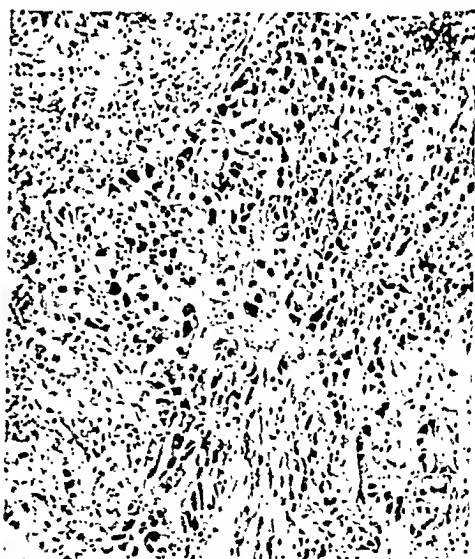


FIG. 357.—*Case 31.* Microscopic appearances of growth.

quadrant, attached to the skin. No glands felt. Radium treatment on March 30, 1928, 54 mgrm. being applied beneath the breast for eleven days. Some contraction followed, and a hard mass remained five months later, which probably consisted of fibrosis only. A second treatment, however, was given on Sept. 4, 1928. Further

contraction followed this, and there was considerable interference with the movements of the right arm. No recurrence took place locally, but the patient died on May 10, 1931, of a malignant ovarian tumour. (*Figs. 354-357.*)

It will be noticed that all these patients were in an advanced stage of the disease. In two the primary growth was big and enlarged axillary glands were present; in one, although no glands could be felt, the tumour was very large. Probably, therefore, special efforts were made to deal with the disease, and in the second and third instances the fibrous contraction which followed the treatment may have been misinterpreted.

### ACCIDENTS.

A theoretical objection to radium treatment in the minds of some people has been the supposed risk of injury to important structures, but, assuming that the operator possesses adequate knowledge and exercises due care, this risk appears to have been somewhat exaggerated. Needles should always be implanted with regard to the known position of vessels, nerves, etc., and no force should be used. If they are entering the right plane, very little resistance is to be met; if resistance is encountered, the needle should be withdrawn and re-inserted. Among the 171 patients I have treated no one has suffered injury to a vessel or a nerve in the axilla. In one patient (*Case 1*) a needle inserted above the clavicle appeared to have injured the transverse cervical artery, which had to be ligatured; unfortunately the needle was not being removed by myself, but by an assistant. In one patient a needle inserted above the clavicle appears to have touched a root of the brachial plexus, so that the patient has complained of some tingling at the point of her thumb. In the same patient the point of a needle below the clavicle momentarily pierced the lung during removal. This was owing to the fact that it had been inserted from without inwards instead of from the opposite direction, so that the point of the needle became unduly depressed during removal. No ill effects followed.

The needles in the intercostal spaces can undoubtedly be of great danger if improperly inserted. On one occasion a needle put in the fourth space caused a local pleurisy and had to be removed. I have had no other accident, but I know of two or three instances in which patients have lost their lives owing to the needles having been placed too deeply, or to their having been too long, so that pressure on their outer ends caused them to enter the mediastinum farther than was intended. In these cases the needles pierced the pericardium or the heart wall, but this should not occur if the details of technique which I have described are closely observed.

### COMBINATION OF RADIUM WITH SURGERY.

Throughout this investigation I have kept before me as the ultimate object to be attained *an improvement in the final results of treatment*, and not the elimination of surgery in favour of radium simply for the sake of eliminating surgery. I have tried to avoid all bias in either direction and to remember that in the end the best results may be obtained by radium alone, by surgery

alone, or by a combination of the two. Each case has therefore been judged on its own merits, and the consequence has been a considerable degree of elasticity in the precise method used.

Obviously there are several ways in which radium may be combined with surgery, and I have not hesitated to use any of them as circumstances seemed to demand.

1. A tumour may be excised locally before any radium treatment is given, in order to remove the greater part of the growth or to establish the diagnosis. An alternative to the removal of a tumour locally before treatment is the local removal of the mammary gland. This may be necessary if the patient's psychology demands that the tumour should be got rid of completely and at once. In either case the excision should be done if possible with the diathermic needle, in order to eliminate as far as possible any dissemination of carcinoma cells. As the axilla is not being dissected, the lymphatics from the breast to the axillary glands will have to be divided, and it is certainly better to do this with the diathermic needle than with the scalpel. If the tumour is small and clearly in an early stage, this procedure seems to be justified by results. Radium treatment has been given after excision, allowing an interval of seven days or more for healing to take place. The treatment is given to the breast and lymphatic areas in exactly the same way as if the tumour were still there. Twelve patients have been treated by local excision of the tumour, of whom ten are at present without signs of disease for periods up to two years. Two have died, but in one of these the growth, as was seen in the section, had, in spite of its small size, already invaded blood-vessels, so that metastases were inevitable. In only one patient, the first treated in this way, has local recurrence taken place, but this patient was inadequately treated, as it now seems to me.

*Case 52.*—Age 42. Lump noticed in left breast for three months. On examination, above the areola near the edge of the mammary gland was a thickened area, 2 cm. in diameter, somewhat hard. No enlarged glands detected. Lump excised on Jan. 28, 1929. Section showed spheroidal-celled carcinoma. Radium treatment to breast and lymphatic areas. Patient has remained well since that time (2 years, 7 months).

*Case 55.*—Age 39. Small lump noticed in right breast for one month. On examination, small mobile lump, 2 cm. diameter, in outer part of right breast. No enlarged glands detected. Radium treatment to breast and lymphatic areas on March 7, 1929. Patient has remained without signs of disease since (2 years, 6 months).

*Case 63.*—Age 58. Small lump removed from left breast by Dr. Bullen, of Lincoln, on April 8, 1929. Section showed spheroidal-celled carcinoma. Radium treatment on May 13, 1929, to breast and lymphatic areas (after removal of large toxic adenoma of thyroid). Patient has remained without signs of disease since (2 years, 4 months).

*Case 85.*—Age 47. A small lump was removed from the inner upper quadrant of the right breast on June 1, 1929, by Mr. Joyce, of Reading. Section showed a scirrhus carcinoma. On examination the patient showed a small scar over the breast, but no signs of active disease. Prophylactic radium treatment to breast and lymphatic areas on Sept. 26, 1929. The patient has remained without signs of disease since (1 year, 11 months).

*Case 90.*—Age 44. Small lump removed from right breast by Dr. Nash-Wortham, of Dorchester. Proved by section to be carcinoma. No glands palpable. Prophylactic radium treatment to breast and lymphatic areas on Nov. 14, 1929. The patient has remained without signs of disease since (1 year, 10 months).

*Case 117.*—Age 59. Small lump in right breast excised in March, 1930, by Dr. Newman, of Bishop's Stortford. Section showed duct carcinoma. Palpable gland in axilla. Prophylactic radium treatment to breast and lymphatic areas on May 16, 1930. Patient has remained without sign of disease since (1 year, 3 months).

*Case 121.*—Age 45. Small lump removed from right breast by Mr. McAdam Eccles on April 1, 1930. Section showed spheroidal-celled carcinoma. Prophylactic radium treatment to breast and lymphatic areas given on June 11. Patient has remained quite well since (1 year, 3 months).

*Case 133.*—Age 45. Lump noticed in right breast for six months. On examination, hard lump in upper outer quadrant,  $3 \times 2$  cm., not attached to skin. Diffuse mastitis in both breasts. Excision of lump on Sept. 16, 1930. Section showed spheroidal-celled carcinoma. Prophylactic radium treatment to breast and lymphatic areas on Oct. 3. Patient has remained quite well since (11 months).

*Case 131.*—Age 46. Small lump noticed in right breast for three weeks, which was removed locally on July 10, 1930. Section showed duct carcinoma. Prophylactic radium treatment to breast and lymphatic areas on Sept. 6. Patient has remained quite well since (1 year).

*Case 93.*—Age 63. Lump noticed in left breast for two months. Soft lump in upper outer quadrant excised by Dr. C. T. Neve, of Croydon, on Nov. 8, 1929. Section showed spheroidal carcinoma. Prophylactic radium treatment to breast and lymphatic areas on Dec. 5. Patient has remained without signs of disease since (1 year, 10 months).



FIG. 358.—Case 48. Position of radium needles.

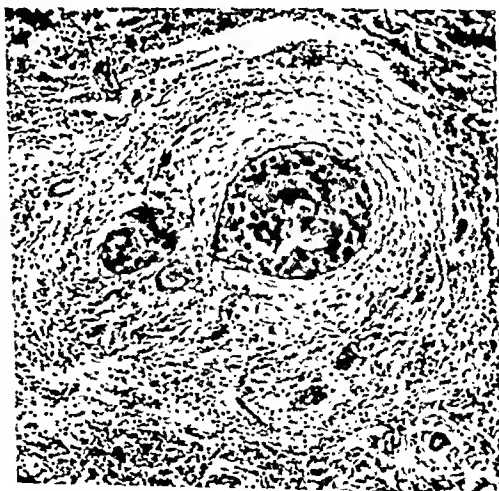


FIG. 359.—Case 48. Section showing invasion of blood-vessels.

*Case 48.*—Age 54. Small swelling noticed in right breast for one month. Enlarged gland noticed in right axilla previously. Lump and gland excised on Nov. 25, 1928. Both proved by section to be carcinoma. Section of primary growth showed invasion of blood-vessels. Insertion of radium on Dec. 28. Patient



remained well until July 16, 1929, when some enlarged glands were felt above the clavicle. Radium inserted above clavicle on Aug. 29. No trace of glands detected on Nov. 5. Patient died of spinal metastases in May, 1930. (*Figs. 358, 359.*),

2. A breast may be removed in whole or in part after treatment for more than one reason. Reference has already been made to this in describing the histological evidence of the effect of radium, and it was there mentioned that the chief reason for removal after treatment is the presence of a residual tumour and consequent uncertainty as to the complete disappearance of the growth. Another reason may be the recurrence of growth after treatment, when excision may be preferred to a second treatment with radium. In some the operation has been proved to be unnecessary, in others some active carcinoma has been found. Among 18 patients treated in this way 16 are alive up to periods of six and a half years, and 15 have no discoverable signs of disease, so that local excision after radium treatment may prove to be a valuable modification which should be practised more freely.

3. Lastly radium may be used as a form of prophylactic treatment after the performance of the radical operation. The radium can be used both in the area of operation and in the situations out of reach of operation, so that it can certainly be made a useful adjuvant in this way. The radium can be inserted at the time of the operation, as has been practised by Mr. Sampson Handley and others, or it may be used after healing has taken place. Three patients whom I have treated in this way have all remained quite well for periods up to two and a half years. I believe that if the radical operation is to continue, as it is likely to do, in frequent use, it may be advantageous to give prophylactic radium treatment whenever possible; the evidence on this point is still incomplete, though Sampson Handley has stated that an improvement in his results has followed his use of radium.

### STIMULATION OF METASTASES.

It has been suggested by some critics of this method of treating carcinoma of the breast that it has stimulated dissemination of the disease, and that metastases have occurred more rapidly than if the patient had not been treated. It is obviously an exceedingly difficult thing to prove or disprove the truth of this. Certainly in the present series a number of patients have developed metastatic growths with disconcerting rapidity. I am unable to assert, however, that this was influenced by the treatment. Practically all of them were in so advanced a stage of the disease that metastases were to be expected whatever form of treatment was used. My impression is that 'follow-up' work among patients who have been operated upon provides quite as many disappointments from the same cause, and that no greater incidence can be attributed to radium. If the incidence were greater, it would presumably be shown in an inferior survival-rate, but as the survival-rate is found to be at least as good with radium (p. 479), the stimulation of metastases seems to be improbable. Theoretically also this effect appears to be unlikely, since experimental work on the effect of radium on living cells seems to show that it acts partly by inhibition of cell division and activity. It is believed also to lessen the blood-supply to the tissues treated.

## CONCLUSIONS.

Experience of interstitial radium treatment of primary carcinoma of the breast in 171 cases over a period of seven years suggests that the following conclusions may be drawn :—

1. In general the results of radium treatment compare favourably with those obtained by any other form of treatment, such as pure surgery. In the most successful cases the patients are virtually normal women, and their expectation of life is at least as great as if they had been subjected to a mutilating operation.

2. No exaggerated claim is made for radium treatment, as that it should be used to supplant surgery. It is claimed, however, that radium ought to have a definite place in the treatment of carcinoma of the breast, and should often be used instead of, or combined with, surgery, according to individual circumstances.

3. The place of radium in the treatment will vary according to the stage of the disease and with other circumstances.

*a.* For very advanced or for inoperable tumours radium treatment is the treatment of choice. Remarkably good results can sometimes be obtained in apparently hopeless conditions.

*b.* For the average intermediate operable tumour the psychology of the patient or other circumstances may sometimes demand the orthodox radical operation. This may be followed by prophylactic radium treatment if desired. For unprejudiced patients the radical operation is seldom necessary, since it is justifiable in most cases to use radium alone, or radium combined with a modified operation. If primary radium treatment is not entirely successful, the patient's prospects are not impaired, and conservative surgery can usually still be applied. An exception is made in the case of very adipose patients, who are probably unsuitable for radium.

*c.* For the earliest and smallest tumours the radical operation is unnecessary. Excellent results can be obtained by radium alone, or by radium combined with the most conservative surgery.

4. The evidence on which these conclusions are based consists of :—

*a.* Preliminary microscopic examination of the tumour in the first fifty and some subsequent patients.

*b.* Microscopic examination of specimens removed for various reasons after radium treatment.

*c.* Systematic clinical examination of all patients after treatment since it was first instituted up to the present time.

*d.* The occurrence of normal pregnancy and lactation in one patient after radium treatment of a fully proved carcinoma of the breast without any recurrence of the disease.

5. Radium may be a very dangerous and inefficient weapon if used with insufficient care or knowledge. The present series of cases has not been without disasters, though these have been due to lack of experience in attaining the correct dosage rather than to technical faults. With proper care and knowledge interstitial radium treatment has no serious dangers.

## ANALYSIS OF 171 CASES OF CANCER OF BREAST TREATED BY RADIUM.

N.S.D. = No signs of disease. N.A.D. = No active disease. B + = Microscopical section positive before treatment. A + or A - = Microscopical section positive or negative after treatment.

No.	AGE	HISTORY	TYPE OF TUMOUR	MICROSCOPICAL SECTION	SIZE OF TUMOUR	GLANDS	TREATMENT	TIME	RESULT TO 15.9.31
1	47	3 years	Contracting, adherent, ulcerated, Inoperable	B +	3 × 4 cm.	-	1.8.24 Ra. breast: 52.8 mgrm. 24 hours 4.12.24 Ra. breast: 11.5 mgrm. 120 hours 2.7.25 Local excision 18.1.27 Ra. breast: 9.0 mgrm. L. areas: 28.5 mgrm. 120 hours 10.1.30 Ra. breast: 10 mgrm. 168 hours	7 years 1 month	N.A.D.
2	59	3 years	Contracting, adherent, skin infiltrated. Inoperable	B +	3 cm.	-	29.1.25 Ra. breast: 20.5 mgrm. 168 hours 26.6.25 Local excision 6.7.26 Ra. breast: 19 mgrm. L. areas: 42 mgrm. 168 hours	4 years 3 months	Died Recurrence in breast, glands, and spine
3	43	2 years	Contracting, adherent, skin infiltrated. Inoperable	B +	5 × 2.5 cm.	+	5.5.25 Ra. breast: 24.5 mgrm. L. areas: 12.5 mgrm. 192 hours 12.11.27 Ra. L. areas: 15 mgrm. 144 hours	6 years 4 months	N.S.D.
4	57	5 months	Massive, adherent, skin nodules. Inoperable	B +	Whole breast	+	1.5.25 Ra. breast: 48 mgrm. L. areas: 8 mgrm. 168 hours 14.5.25 X-ray treatment 23.10.25 Local excision	7 months	Died Thoracic metastases
5	50	7 months	Massive, adherent. Inoperable	B +	Whole breast	+	11.6.25 Ra. breast: 36 mgrm. L. areas, 11 mgrm. 168 hours 23.6.25 X-ray treatment	2 months	Died Metastases in liver
6	35	1 year	Massive, fungating. Inoperable	B +	Whole breast	+	10.7.25 Ra. breast: 20 mgrm. L. areas: 10.5 mgrm. 168 hours	5 months	Died Thoracic metastases

7	57	5 years	Massive, skin involved, Inoperable	B +	Whole breast	+	12.8.25 Ra. breast : 72 mgrm. L. areas : 22.5 mgrm. 168 hours 12.3.26 Ra. breast : 63.5 mgrm. 144 hours	9 months	Died Thoracic meta- stases
8	57 (mule)	1 year	Contracting, fixed, skin involved. Operable	B +	2.5 cm.	+	23.3.26 Ra. breast : 64 mgrm. L. areas : 25 mgrm. 148 hours	1 year 7 months	Died Intercurrent disease. No local recurrence
9	60	0 months	Small. Operable	B + A -	2 cm.	-	18.5.26 Lump excised Ra. breast : 42 mgrm. L. areas : 14 mgrm. 148 hours 4.4.27 Ra. breast : 20.5 mgrm. 144 hours 23.9.27 Local excision 7.12.28 Ra. L. areas, 24 mgrm. 168 hours	4 years 5 months	Died Thoracic meta- stases
10	63	3 years	Massive, adherent, skin infiltrated. Inoperable	B +	7.5 x 5 cm.	++	13.7.26 Ra. breast : 57.0 mgrm. L. areas : 39.5 mgrm. 148 hours 3.5.27 Ra. breast : 22.5 mgrm. 144 hours 17.1.28 Ra. breast : 33 mgrm. 120 hours 7.6.29 Ra. breast : 18 mgrm. 168 hours 9.10.30 Ra. surface application	5 years 2 months	Disease arrested
11	49	18 months	Contracting, adherent, skin infiltrated. Inoperable	B +		-	6.8.26 Ra. breast : 60.5 mgrm. L. areas : 20.0 mgrm. 130 hours	5 years 1 month	No disease locally. Spinal metastases
12	47	12 years	Contracting, ulcerated, adherent. Inoperable	B +	5 cm.	+	19.11.26 Ra. breast : 51.5 mgrm. L. areas : 37.5 mgrm. 168 hours 20.7.28 Ra. breast : 22 mgrm. 168 hours 25.10.29 Ra. gland on other side : 18 mgrm. 168 hours	4 years 6 months	Died Metastases in skin, media- stinum
13	51	11 months	Massive, skin dimpled. Metastases in mediastinum and skull. Operable	B + A +	7 x 8 cm.	+	21.12.26 Ra. breast : 45 mgrm. L. areas : 28.5 mgrm. 168 hours 29.9.27 Ra. breast : 10 mgrm. L. areas : 2 mgrm. 120 hours 19.1.28 Local excision	1 year 2 months	Died Septicemia

ANALYSIS OF 171 CASES OF CANCER OF BREAST TREATED BY RADIUM—continued.

No.	AGE	HISTORY	TYPE OF TUMOUR	MICRO-SCOPICAL SECTION	SIZE OF TUMOUR	GLANDS	TREATMENT	TIME	RESULT TO 15.9.31
14	56	10 years	Massive, adherent, skin infiltrated. Inoperable	B + A +	5 × 4 cm.	—	10.2.27 Ra. breast: 32 mgrm. L. areas: 7 mgrm. 144 hours 8.6.27 Ra. breast: 36 mgrm. 144 hours 1.11.27 Local excision	4 years 2 months	Died No disease locally Spinal meta- stases
15	40	1 year	Adherent to skin and nipple. Operable	B +	2.5 × 3 cm.	+	17.2.27 Ra. breast: 45 mgrm. L. areas: 32.5 mgrm. 144 hours	4 years 6 months	N.S.D.
16	62	4 months	Skin attached. Operable	B +	6 cm.	+	14.4.27 Ra. breast: 51.5 mgrm. L. areas: 45 mgrm. 168 hours 12.4.28 Ra. breast: 48 mgrm. 192 hours	3 years 9 months	Died Late necrosis Mediastinal metastases
17	54	9 months	Massive, ulcerated, adherent. Inoperable	B +	8 × 5 cm.	+	20.5.27 Ra. breast: 39 mgrm. L. areas: 21 mgrm. 168 hours	9 months	Died Mediastinal metastases
18	46	2 months	Massive, adherent, skin infiltrated. Inoperable	B +	10 cm.	+	14.6.27 Ra. breast: 58.5 mgrm. L. areas: 35.5 mgrm. 168 hours 3.2.28 Ra. breast: 19 mgrm. L. areas: 16 mgrm. 192 hours	11 months	Died Spinal meta- stases
19	70	6 years	Contracting, skin infiltrated. Inoperable	B +	2 × 1.5 cm.	+	2.8.27 Ra. breast: 36 mgrm. L. areas: 40.5 mgrm. 168 hours	4 years 1 month	N.S.D.
20	42	18 months	Adherent, skin infiltrated. Inoperable	B +	4 cm.	+	9.8.27 Ra. breast: 27 mgrm. L. areas: 41.5 mgrm. 168 hours 31.1.28 Ra. breast: 60 mgrm. 168 hours 20.7.28 X-ray treatment	2 years 2 months	Died General meta- stases
21	61	3 years	Ulcerated. Operable	B + A +	2 × 3 cm.	—	20.9.27 Ra. breast: 20 mgrm. L. areas: 28.5 mgrm. 168 hours 31.1.28 Local excision 5.12.30 Excision in situ	3 years 11 months	N.S.D.

# RADIUM TREATMENT OF BREAST CANCER 463

61	5 months	Attached to skin. Operable	B + A -	3 cm.	25.10.27	Ra. breast : 54.5 mgrm. L. areas : 27.0 mgrm. 168 hours	3 years 10 months	N.S.D.
23	44	2 years	B +	3 cm.	28.6.20	Ra. to breast : 41 mgrm. 168 hours		
24	48	2 years	B +	5 × 2.5 cm.	19.12.29 4.7.30	X-ray treatment Local excision		
25	51	2 years	B +	5 cm.	25.10.27	Ra. breast : 30 mgrm. L. areas : 32.5 mgrm. 163 hours	3 years 10 months	N.S.D.
26	30	9 months	B +	5 cm.	4.11.27	Ra. breast : 33 mgrm. L. areas : 19.5 mgrm. 120 hours	3 years 10 months	N.S.D.
27	62	5 years	B +	5 × 4 cm.	30.11.27	Ra. breast : 39 mgrm. L. areas : 38.5 mgrm. 144 hours	8 months	Died Skeletal meta- stases
28	57	2½ years	B +	7 cm.	16.12.27	Ra. breast : 31 mgrm. L. areas : 32 mgrm. 168 hours	3 years 8 months	Pulmonary metastases
29	51	4 years	B +	4 × 3 cm.	12.4.28 20.11.29	Ra. breast : 26 mgrm. 14½ hours Ra. axilla : 17 mgrm. 168 hours		
30	51	1 month	B +	5 cm.	16.12.27	Ra. breast : 43 mgrm L. areas : 35.5 mgrm. 144 hours	3 years 8 months	N.S.D.
			B +	10 × 8 cm.	3.10.30	Ra. breast : 2½ mgrm. Axilla : 6 mgrm. 168 hours		
			B +	8 cm.	6.1.28	Ra. breast : 32 mgrm. L. areas : 64.5 mgrm. 168 hours	1 year	Died Disappearance of tumour. Spinal metastases
			B +	5 cm.	1926, 1927 10.2.28	X-ray treatment Ra. breast : 95 mgrm. L. areas : 35 mgrm. 168 hours	10 months	Died Skeletal meta- stases
			B +		17.2.28	Ra. breast : 42 mgrm. L. areas : 35 mgrm. 168 hours	2 years 9 months	Died Thoracic metastases
			B +		5.7.29	Ra. axilla : 33 mgrm. 168 hours		

## ANALYSIS OF 171 CASES OF CANCER OF BREAST TREATED BY RADIUM—continued.

No.	AGE	HISTORY	TYPE OF TUMOUR	MICRO-SCOPICAL SECTIONS	SIZE OF TUMOUR	GLANDS	TREATMENT	TIME	RESULT TO 12.31.31
31	58	15 months	Large, skin adherent. Operable	B +	6 cm.	—	30.3.28 Ra. breast: 54 mgrm. L. areas: 38 mgrm. 264 hours 4.9.28 Ra. breast: 46 mgrm. 168 hours	3 years 1 month	Died Malignant ovarian tumour
32	47	9 months	Attached to skin. Operable	B +	4 cm.	+	18.4.28 Ra. breast: 30.5 mgrm. L. areas: 30 mgrm. 216 hours 18.1.29 Ra. breast: 15 mgrm. 144 hours 7.5.29 X-ray treatment	2 years 5 months	Died Visceral meta- stases
33	55	7 months	Attached to skin. Operable	B +	3.5 cm.	+	20.4.28 Ra. breast: 32 mgrm. L. areas: 26 mgrm. 168 hours 1.11.29 Ra. breast: 29 mgrm. L. areas: 12 mgrm. 168 hours	1 year 11 months	Died Mediastinal metastases
34	68	2 years	Contracting, massive, adherent. Inoperable	B +	7 cm.	+	27.4.28 Ra. breast: 50 mgrm. L. areas: 26.5 mgrm. 160 hours 4.1.29 Ra. breast: 36 mgrm. 168 hours	2 years	Died No local recur- rence. Spinal metastases
35	55	2 months	Massive. Operable	B +	10 × 7 cm.	—	27.4.28 Ra. breast: 36 mgrm. L. areas: 29 mgrm. 240 hours	11 months	Died Skeletal meta- stases
36	68	1 year	Contracting, ulcerated, adherent. Inoperable	B +	4 × 5 cm.	—	11.5.28 Ra. breast: 29 mgrm. L. areas: 27.5 mgrm. 168 hours 11.4.30 Ra. breast: 10 mgrm. 168 hours	3 years 4 months	N.A.D.
37	48	9 months	Contracting, infiltration of skin. Operable	B +	4 cm.	+	18.5.28 Ra. breast: 46 mgrm. L. areas: 30 mgrm. 168 hours 17.11.29 Ra. axilla: 21 mgrm. 168 hours	2 years	Died Metastases in jaw and spine

# RADIUM TREATMENT OF BREAST CANCER 465

38	77	1 year	Massive, skin attached. Operable	B + A -	7 cm.	-	1.6.28 Ra. breast : 47 mgrm. L. areas : 27 mgrm. 168 hours 7.12.28 Excision of residual lump	3 years 3 months	N.S.D.
39	52	1 year	Massive, adherent. Inoperable	B +	10 cm.	-	8.6.28 Ra. breast : 63 mgrm. L. areas : 28 mgrm. 240 hours 9.4.29 Ra. breast : 30 mgrm. 120 hours	1 year 6 months	Died Mediastinal metastases
40	57	2 months	Adherent. Inoperable	B +	6 x 3.5 cm.	-	8.6.28 Ra. breast : 40 mgrm. L. areas : 28 mgrm. 168 hours	3 years 3 months	N.S.D.
41	68	2 years	Massive, fungating, Inoperable	B + A +	6 cm.	+	22.6.28 Ra. breast : 42 mgrm. L. areas : 28.5 mgrm. 168 hours 8.3.29 Excision of residual lump	9 months	Died Intercurrent dis- ease
42	58	2 years 6 months	Contracting, adherent. Operable	B + A +	6 cm.	+	22.6.28 Ra. breast : 43 mgrm. L. areas : 26 mgrm. 168 hours 1.8.29 Ra. Pect. muse. : 24 mgrm. 168 hours 21.11.29 Local excision : breast	3 years 2 months	N.S.D.
43	63	3 years	Ulcerated. Operable	B +	5 cm.	-	6.7.28 Ra. breast : 42 mgrm. L. areas : 24 mgrm. 168 hours	2 years 5 months	Died Pulmonary metastases
44	69	1 year	Massive, ulcerated. Operable	B +	5 x 8 cm.	+	18.7.28 Ra. breast : 60 mgrm. L. areas : 34 mgrm. 144 hours	7 months	Died Pulmonary metastases
45	40	2 years	Contracting, ulcerated, adherent. Operable	B +	5 x 6 cm.	+	23.8.28 Ra. breast : 50.5 mgrm. L. areas : 26 mgrm. 264 hours 15.2.29 Ra. breast : 52 mgrm. L. areas : 6 mgrm. 168 hours 29.8.29 X-ray treatment	1 year 8 months	Died Thoracic and spinal meta- stases
46	59	2 months	Attached to skin. Operable	B +	5 x 6 cm.	-	14.9.28 Ra. Lt. breast : 43 mgrm. L. areas : 23 mgrm. 168 hours 10.5.29 Ra. axilla : 30 mgrm. 168 hours 24.4.31 Removal, Rt. breast and insertion of 66 mgrm. Ra. 168 hours	3 years	N.S.D.



## ANALYSIS OF 171 CASES OF CANCER OF BREAST TREATED BY RADIUM—continued.

No.	AGE	HISTORY	TYPE OF TUMOUR	MICROSCOPICAL SECTION	SIZE OF TUMOUR	GLANDS	TREATMENT	TIME	RESULT TO 15.9.31
47	68	9 months	Contracting, ulcerated, adherent. Inoperable	B +	6.5 × 5 cm.	—	23.11.28 Ra. breast : 36 mgrm. L. areas : 20 mgrm. 144 hours 6.12.29 X-ray treatment	2 years 2 months	Died Thoracic meta- stases
48	54	3 months	Small. Operable. Microscopic invasion of blood-vessels	B +	2 cm.	+	25.11.28 Lump and glands excised 28.12.28 Ra. breast : 27 mgrm. L. areas : 37.5 mgrm. 168 hours 29.8.29 Ra. L. areas : 10 mgrm. 168 hours	1 year 6 months	Died Spinal meta- stases
49	61	1 year	Attached to skin. Operable		4.5 cm.	+	4.1.29 Ra. breast : 51 mgrm. L. areas : 34 mgrm. 168 hours 10.10.29 Ra. breast : 29 mgrm. L. areas : 7 mgrm. 168 hours	1 year 4 months	Died Mediastinal metastases
50	39	1 year	Attached to skin. Operable		5 × 4 cm.	—	5.2.29 Ra. breast : 33 mgrm. L. areas : 30 mgrm. 90 hours 5.8.29 Ra. breast : 46 mgrm. 168 hours	1 year 2 months	Died Mediastinal metastases
51	43	2 months	Attached to nipple. Operable	A +	3 cm.	+	9.2.29 Ra. breast : 30 mgrm. L. areas : 30 mgrm. 168 hours 19.9.30 Ra. breast : 85 mgrm. 168 hours 19.6.31 Removal of breast, and deep X-ray treatment	2 years 7 months	Disease arrested
52	42	3 months	Early. Operable	B +	2 cm.	—	28.1.29 Lump excised 13.2.29 Ra. breast : 27 mgrm. L. areas : 21 mgrm. 120 hours	2 years 7 months	N.S.D.
53	46	1 year	Attached to skin. Operable	A +	4 cm.	+	20.2.29 Ra. breast : 48 mgrm. L. areas : 26 mgrm. 144 hours 24.9.29 Local removal of breast 24.10.29 X-ray treatment	1 year 4 months	Died Thoracic meta- stases

# RADIUM TREATMENT OF BREAST CANCER 467

54	37	4 months	Attached to nipple. Operable	A +	5 × 3 cm.	+	1.3.29 Ra. breast : 36 mgrm. L. areas : 26 mgrm. 10.7.30 Local removal of breast	2 years 6 months	N.S.D.
55	39	1 month	Early. Operable	B +	2 cm.	—	27.2.29 Excision of lump 7.3.29 Ra. breast : 30 mgrm. L. areas : 42 mgrm. 144 hours	2 years 6 months	N.S.D.
56	41	7 months	Massive, adherent, skin infiltrated. Inoperable		Whole breast	++	8.3.29 Ra. breast : 48 mgrm. L. areas : 42 mgrm. 144 hours	3 months	Died of the dis- ease
57	65	Local removal of breast, 31.12.28		B +		—	13.3.29 Ra. 41 mgrm. 168 hours	2 years 6 months	N.S.D.
58	47	8 months	Adherent. Operable	A +	6 × 2.5 cm.	+	21.3.29 Ra. breast : 28 mgrm. L. areas : 26 mgrm. 168 hours	2 years 6 months	N.S.D.
59	63	2 weeks	Early, skin attached. Operable				7.2.30 Excision of residual lump 16.1.31 Ra. nodule : 12 mgrm. 168 hours	2 years 5 months	Metastases in femur
60	71	1 year	Contracting, adherent. Operable		2.5 cm.	—	9.4.29 Ra. breast : 30 mgrm. L. areas : 26 mgrm. 138 hours	2 years 5 months	N.S.D.
61	49	3 months	Not adherent. Operable		2.5 cm.	—	19.4.29 Ra. breast : 25 mgrm. L. areas : 28.5 mgrm. 120 hours	2 years 4 months	N.A.D.
62	58	6 months	Skin attached. Operable		5 cm.	—	3.5.29 Ra. breast : 47 mgrm. L. areas : 20 mgrm. 168 hours	2 years 4 months	N.S.D.
63	58	Lump excised from breast, 8.4.29		+			3.5.29 Ra. breast : 36 mgrm. L. areas : 18.5 mgrm. 168 hours	2 years 4 months	N.S.D.
64	58	1 year	Contracting, skin infil- trated. Operable	A +	5 cm.	—	13.5.29 Ra. breast : 27 mgrm. L. areas : 26 mgrm. 136 hours	2 years 4 months	N.S.D.
							16.5.29 Ra. breast : 36 mgrm. L. areas : 21 mgrm. 168 hours	2 years 4 months	N.S.D.
							3.7.31 Removal of breast and axillary glands	2 years 3 months	Disease arrested

## ANALYSIS OF 171 CASES OF CANCER OF BREAST TREATED BY RADIUM—continued.

No.	AGE	HISTORY	TYPE OF TUMOUR	MICROSCOPICAL SECTION	SIZE OF TUMOUR	GLANDS	TREATMENT	TIME	RESULT TO 15.9.31
47	68	9 months	Contracting, ulcerated, adherent. Inoperable	B +	6.5 × 5 cm.	—	23.11.28 Ra. breast : 36 mgrm. L. areas : 20 mgrm. 144 hours 6.12.29 X-ray treatment	2 years 2 months	Died Thoracic meta- stases
48	54	3 months	Small. Operable. Microscopic invasion of blood-vessels	B +	2 cm.	+	25.11.28 Lump and glands excised 28.12.28 Ra. breast : 27 mgrm. L. areas : 37.5 mgrm. 168 hours 29.8.29 Ra. L. areas : 10 mgrm. 168 hours	1 year 6 months	Died Spinal meta- stases
49	61	1 year	Attached to skin. Operable		4.5 cm.	+	4.1.29 Ra. breast : 51 mgrm. L. areas : 34 mgrm. 168 hours 10.10.29 Ra. breast : 29 mgrm. L. areas : 7 mgrm. 168 hours	1 year 4 months	Died Mediastinal metastases
50	39	1 year	Attached to skin. Operable		5 × 4 cm.	—	5.2.29 Ra. breast : 33 mgrm. L. areas : 30 mgrm. 90 hours 5.8.29 Ra. breast : 46 mgrm. 168 hours	1 year 2 months	Died Mediastinal metastases
51	43	2 months	Attached to nipple. Operable	A +	3 cm.	+	9.2.29 Ra. breast : 30 mgrm. L. areas : 30 mgrm. 168 hours 19.9.30 Ra. breast : 85 mgrm. 168 hours 19.6.31 Removal of breast, and deep X-ray treatment	2 years 7 months	Disease arrested
52	42	3 months	Early. Operable	B +	2 cm.	—	28.1.29 Lump excised 13.2.29 Ra. breast : 27 mgrm. L. areas : 21 mgrm. 120 hours	2 years 7 months	N.S.D.
53	46	1 year	Attached to skin. Operable	A +	4 cm.	+	20.2.29 Ra. breast : 48 mgrm. L. areas : 26 mgrm. 144 hours 24.9.29 Local removal of breast 24.10.29 X-ray treatment	1 year 4 months	Died Thoracic meta- stases

# RADIUM TREATMENT OF BREAST CANCER 467

54	57	4 months	Attached to nipple. Operable	A +	5 × 3 cm.	+	1.3.29 Ra. breast : 36 mgrm. L. areas : 26 mgrm. 168 hours Local removal of breast	2 years 6 months	N.S.D.
55	39	1 month	Early. Operable	B +	2 cm.	-	10.7.30 Excision of lump Ra. breast : 30 mgrm. L. areas : 26 mgrm. 144 hours	2 years 6 months	N.S.D.
56	41	7 months	Massive, adherent, skin infiltrated. Inoperable		Whole breast	++	27.2.29 Excision of lump 7.3.29 Ra. breast : 48 mgrm. L. areas : 42 mgrm. 144 hours	3 months	Died of the dis- ease
57	65	Local removal of breast, 31.12.28		B +		-	8.3.29 Ra. breast : 41 mgrm. 168 hours	2 years 6 months	N.S.D.
58	47	8 months	Adherent. Operable	A +	6 × 2.5 cm.	+	21.3.29 Ra. breast : 28 mgrm. L. areas : 26 mgrm. 168 hours	2 years 6 months	N.S.D.
59	63	2 weeks	Early, skin attached. Operable				7.2.30 Excision of residual lump 16.1.31 Ra. nodule : 12 mgrm. 168 hours	2 years 5 months	Metastases in femur
60	71	1 year	Contracting, adherent. Operable		2.5 cm.	-	9.4.29 Ra. breast : 30 mgrm. L. areas : 26 mgrm. 138 hours	2 years 5 months	N.S.D.
61	49	3 months	Not adherent. Operable		2.5 cm.	-	19.4.29 Ra. breast : 25 mgrm. L. areas : 28.5 mgrm. 120 hours	2 years 4 months	N.A.D.
62	58	6 months	Skin attached. Operable		5 cm.	-	3.5.29 Ra. breast : 47 mgrm. L. areas : 20 mgrm. 108 hours	2 years 4 months	N.S.D.
63	58	Lump excised from breast, 8.4.29		2.5 cm.	+		3.5.29 Ra. breast : 36 mgrm. L. areas : 18.5 mgrm. 168 hours	2 years 4 months	N.S.D.
64	58	1 year	Contracting, skin infil- trated. Operable	B +		-	13.5.29 Ra. breast : 27 mgrm. L. areas : 26 mgrm. 136 hours	2 years 4 months	N.S.D.
				A +	5 cm.	-	16.5.29 Ra. breast : 36 mgrm. L. areas : 21 mgrm. 168 hours	2 years 3 months	Disease arrested
							3.7.31 Removal of breast and axillary glands		

## ANALYSIS OF 171 CASES OF CANCER OF BREAST TREATED BY RADIUM—continued.

No.	AGE	HISTORY	TYPE OF TUMOUR	MICROSCOPICAL SECTION	SIZE OF TUMOUR	GLANDS	TREATMENT	TIME	RESULT TO 15.9.31
65	33	5 weeks	Small. Operable		2.5 cm.	+	31.5.29 Ra. breast : 27 mgrm. L. areas : 32 mgrm. 168 hours 23.9.30 Ra. breast : 28 mgrm. Axilla : 6 mgrm. 168 hours	2 years 1 month	Died Thoracic meta- stases. Carci- noma of uterus
66	57	2 weeks	Early. Operable		1.5 cm.	—	8.6.29 Ra. breast : 20 mgrm. L. areas : 26 mgrm. 168 hours	2 years 3 months	N.S.D.
67	47	6 months	Adherent, ulcerated. Inoperable		5 cm.	—	11.6.29 Ra. breast : 25 mgrm. L. areas : 32 mgrm. 168 hours	5 months	Died Spinal meta- stases
68	52	1 year	Not adherent. Operable		6 cm.	+	12.6.29 Ra. breast : 24 mgrm. L. areas : 21 mgrm. 168 hours	2 years 3 months	N.S.D.
69	71	3½ years	Contracting, skin inflamed. Operable	A Br.— Gl. +	4 cm.	+	14.6.29 Ra. breast : 35 mgrm. L. areas : 26 mgrm. 168 hours 17.10.30 Local removal of breast. L. Ax. glands	2 years 3 months	N.A.D.
70	60	6 weeks	Skin attached. Operable		6 cm.	—	18.6.29 Ra. breast : 42 mgrm. L. areas : 30 mgrm. 168 hours 12.12.29 Ra. L. areas : 15 mgrm. 168 hours 4.9.30 Ra. Surface application	1 year 5 months	Died Visceral meta- stases
71	67	6 weeks	Not adherent. Operable		5 cm.	+	21.6.29 Ra. breast : 31 mgrm. L. areas : 31 mgrm. 168 hours 2.9.30 Ra. breast : 27 mgrm. L. areas : 40 mgrm. 168 hours	2 years 2 months	N.S.D.
72	62	9 months	Massive, fungating. Inoperable		12.5 cm.	+	26.6.29 Ra. breast : 32.1 mgrm. L. areas : 30.1 mgrm. 168 hours	2 months	Died Visceral meta- stases

# RADIUM TREATMENT OF BREAST CANCER 469

73	44	2-3 years	Skin attached. Operable		5 cm.	-	20.6.29 Ra. breast : 26 mgrm. L. areas : 19.7 mgrm. 168 hours	2 years 2 months	N.S.D.
74	55	Removal	of breast with axillary glands, 23.10.29	B +		+	8.7.29 Ra. L. areas : 32 mgrm. 168 hours	2 years 2 months	N.S.D.
75	60	3 weeks	Skin adherent. Operable	A +	4 cm.	+	11.7.29 Ra. breast : 39 mgrm. L. areas : 24 mgrm. 168 hours 23.4.30 Local removal of breast 19.6.30 Ra. to scar : 40 mgrm. 1.4.4 hours 25.4.31 Surface application Ra. to scar	2 years 2 months	No local recur- rence. Thoracic metastases
76	50		Adherent. Operable	A -	8 cm.	-	19.7.29 Ra. breast : 53 mgrm. L. areas : 31 mgrm. 168 hours 9.1.30 Ra. breast : 14 mgrm. 168 hours 25.11.30 Local removal of breast	2 years 1 month	N.S.D.
77	60	Removal	of breast with axillary glands, 7.6.29	B +		+	21.8.29 Ra. L. areas : 35 mgrm. 1.4.4 hours	2 years	N.S.D.
78	40	6 weeks	Cystic, skin not adher- ent. Operable	A +	4 x 5 cm.	-	18.1.29 Ra. breast : 24 mgrm. L. areas : 23 mgrm. 168 hours 24.9.29 Excision of cyst 10.4.31 Local removal of breast	2 years 7 months	N.S.D.
79	55	6 months	Skin adherent. Operable	A +	6 cm.	-	28.8.29 Ra. breast : 42 mgrm. L. areas : 26 mgrm. 168 hours 1.5.30 Local removal of breast 9.30 Ra. surface application	2 years	Mediastinal metastases. Skin nodules
80	58	8 months	Massive, adherent, skin infiltrated. Inoperable		Whole breast	+	3.9.29 Ra. breast : 38.5 mgrm. L. areas : 22.5 mgrm. 168 hours 12.29 X-ray treatment	8 months	Died Visceral metastases
81	62	1 year	Nipple attached, skin infiltrated. Operable		10 cm.	+	4.9.29 Ra. breast : 58 mgrm. L. areas : 28 mgrm. 168 hours	8 months	Died Metastases in skin and skeleton
82	42	3 weeks	Nipple adherent Operable	A -	7 cm.	-	10.9.29 Ra. breast : 46 mgrm. L. areas : 24.5 mgrm. 168 hours 30.10.30 Local removal of breast	2 years	N.S.D.

## ANALYSIS OF 171 CASES OF CANCER OF BREAST TREATED BY RADIUM—continued.

No.	AGE	HISTORY	TYPE OF TUMOUR	MICRO-SCOPICAL SECTION	SIZE OF TUMOUR	GLANDS	TREATMENT	TIME	RESULT TO 15.3.31
83	58	2 years	Small. Operable		2.5 cm.	+	11.9.29 Ra. breast: 21 mgrm. L. areas: 20 mgrm. 168 hours	2 years	N.S.D.
84	86		Skin attached. Operable		4 × 3 cm.	+	18.9.29 Ra. breast: 30 mgrm. L. areas: 26 mgrm. 168 hours	1 year 11 months	No local disease. Visceral meta- stases
85	47	1.6.29,	lump removed from breast	B +		—	26.9.29 Ra. breast: 24 mgrm. L. areas: 26 mgrm. 168 hours	1 year 11 months	N.S.D.
86	43	10 months	Skin attached. Operable		4 cm.	+	2.10.29 Ra. breast: 38 mgrm. L. areas: 28 mgrm. 192 hours	1 year 2 months	Died Spinal meta- stases
87	49	3 months	Contracting. Operable		5 × 4 cm.	+	3.10.29 Ra. breast: 27 mgrm. L. areas: 33.5 mgrm. 168 hours 1.5.31 Ra. breast: 10 mgrm. L. areas: 14 mgrm. 168 hours	1 year 10 months	Disease arrested
88	63	2 years 8 months	Massive. Operable		10 cm.	+	28.10.29 Ra. breast: 57 mgrm. L. areas: 32 mgrm. 168 hours 13.11.30 Ra. breast: 50 mgrm. 168 hours	1 year 8 months	Died Visceral meta- stases. Local improvement
89	37	6 months	Small, not adherent. Operable		2 × 1.5 cm.	—	1.3.29 Ra. breast: 21 mgrm. L. areas: 20 mgrm. 144 hours	2 years 6 months	N.S.D.
90	44	June, 1929,	lump excised from breast	B +		—	14.11.29 Ra. breast: 36 mgrm. L. areas: 29 mgrm. 168 hours	1 year 10 months	N.S.D.
91	65	5 months	Not adherent. Operable		4 × 5 cm.	+	21.11.29 Ra. breast: 40 mgrm. L. areas: 38 mgrm. 168 hours	3 months	Died Intercurrent disease
92	56	2 years	Ulcerated. Inoperable		6 cm.	—	29.11.29 Ra. breast: 48 mgrm. L. areas: 19 mgrm. 168 hours	1 year	Died Visceral meta- stases

# RADIUM TREATMENT OF BREAST CANCER 471

	93	63	2 months	Early, skin attached. Operable	B +	2 cm.	+	8.11.29 5.12.29	1 year 10 months	N.S.D.
	94	35	4 months	Skin attached. Operable	A Br. - Gl. +	5 cm.	-	Lump excised Ra. breast : 34 mgrm. L. areas : 35 mgrm. 168 hours	1 year 9 months	N.S.D.
	95	75	4 years	Contracting, adherent. Inoperable		5 cm.	-	12.12.29 Ra. breast : 53 mgrm. L. areas : 30 mgrm. 160 hours 12.1.31 Local excision of the residual lump with gland	1 year 9 months	N.S.D.
	96	64	2 years	Massive, skin attached. Operable		6 x 7 cm.	+	13.12.29 Ra. breast : 54 mgrm. L. areas : 26 mgrm. 144 hours	1 year 9 months	N.S.D.
	97	57	1 month	Skin attached. Operable		4 cm.	-	20.12.29 Ra. breast : 56 mgrm. L. areas : 23 mgrm. 168 hours	1 year	Died Spinal meta- stases
	98	73	2 weeks	Early, nipple attached. Operable		4 cm.	-	30.12.29 Ra. breast : 50 mgrm. L. areas : 23 mgrm. 168 hours	1 year 8 months	Metastases in C.N.S.
	99	48	Several months	Skin attached. Operable		4 x 2 cm.	-	9.4.30 Ra. breast : 42 mgrm. L. areas : 24 mgrm. 100 hours	1 year 5 months	N.A.D.
	100	57	9 months	Small, attached to skin. Operable		2.5 cm.	+	23.1.30 Ra. breast : 25 mgrm. L. areas : 32 mgrm. 168 hours	1 year 7 months	N.S.D.
	101	50	3 months	Skin infiltrated, nipple attached. Inoperable		5 cm.	-	24.1.30 Ra. breast : 38 mgrm. L. areas : 32 mgrm. 168 hours	1 year 7 months	N.S.D.
	102	66	3 months	Small. Operable		3 cm.	-	14.2.30 Ra. breast : 47 mgrm. L. areas : 29 mgrm. 168 hours	1 year 7 months	No disease locally. Abdo- minal metastases
	103	41	1 year	Massive, nipple at- tached, secondary growth in the eye. Operable	Whole breast	+	20.2.30 Ra. breast : 31 mgrm. L. areas : 29 mgrm. 168 hours	7 months	Died Metastases in liver	
							21.2.30 Ra. breast : 74 mgrm. L. areas : 32 mgrm. 168 hours	4 months	Died General meta- stases	



ANALYSIS OF 171 CASES OF CANCER OF BREAST TREATED BY RADIUM—*continued*.

No.	AGE	HISTORY	TYPE OF TUMOUR	MICRO-SCOPICAL SECTION	SIZE OF TUMOUR	GLANDS	TREATMENT	TIME	RESULT TO 15.9.31
104	55	5 years	Massive, ulcerated, adherent. Inoperable	A +	13 cm.	+	27.2.30 Ra. breast : 54 mgrm. L. areas : 21 mgrm. 168 hours 27.8.30 Ra. breast : 32 mgrm. 168 hours 2.7.31 Excision of residual mass	1 year 6 months	N.S.D.
105	56	1 week	Small, skin attached. Operable		2 cm.	+	1.3.30 Ra. breast : 24 mgrm. L. areas : 20 mgrm. 168 hours	1 year 6 months	N.S.D.
106	49	7 months	Massive, skin attached. Operable	A +	7 × 3 cm.	—	7.3.30 Ra. breast : 51 mgrm. L. areas : 25 mgrm. 168 hours 29.11.30 Local removal of breast	1 year 6 months	N.S.D.
107	53	3 weeks	Skin attached. Operable	A +	5 cm.	—	6.3.30 Ra. breast : 47 mgrm. L. areas : 30 mgrm. 168 hours 16.10.30 Ra. breast : 71 mgrm. 168 hours 2.7.31 Excision of residual nodule	1 year 6 months	N.S.D.
108	59	7 months	Massive, nipple attached. Operable		6 cm.	—	21.3.30 Ra. breast : 24 mgrm. L. areas : 29 mgrm. 168 hours	7 months	Died Metastases in liver
109	72	6 years	Contracting, skin infiltrated. Operable		4 cm.	—	1.4.30 Ra. breast : 42 mgrm. L. areas : 24 mgrm. 168 hours 17.3.31 Ra. recurrent nodule in skin, 13 mgrm. 168 hours	1 year 5 months	N.A.D.
110	65	1 week	Skin attached. Inoperable		4 cm.	++	8.4.30 Ra. breast : 39 mgrm. L. areas : 20 mgrm. 168 hours	1 year	Died Skeletal metastases
111	50	1 month	Massive, not adherent. Operable	A +	7 × 5 cm.	+	11.4.30 Ra. breast : 40 mgrm. L. areas : 20 mgrm. 192 hours 6.2.31 Local removal of breast	1 year 5 months	N.S.D.

# RADIUM TREATMENT OF BREAST CANCER 473

112	57	1 year	Skin attached, nipple retracted. Operable	5 x 7 cm.	10.4.30 Ra. breast : 42 mgrm. L. areas : 22 mgrm. 23.10.30 Ra. breast : 70 mgrm. 168 hours	1 year 5 months	No local disease. Mass in abdomen and (?) metastases Rt. femur
113	40	4 months	Nipple retracted. Operable	5 x 4 cm.	20.3.30 Ra. breast : 27 mgrm. L. areas : 21 mgrm. 168 hours	1 year 5 months	N.S.D.
114	57	3 months	Not adherent. Operable	Diffuse mass	27.3.30 Local removal of breast Ra. L. areas : 30 mgrm. 168 hours	1 year 5 months	N.S.D.
115	54	6 months	Massive, skin attached. Inoperable	8 x 10 cm.	8.5.30 Ra. breast : 68 mgrm. L. areas : 18 mgrm. 18.9.30 Ra. breast : 54 mgrm. L. areas : 18 mgrm. 168 hours	9 months	Died Metastases
116	66	8 months	Adherent to skin and deep fascia, nipple retracted. Operable	5 cm.	9.5.30 Ra. breast : 30 mgrm. L. areas : 32 mgrm. 168 hours	1 year 4 months	N.S.D.
117	59	March, 1930, small lump excised from breast		+	27.4.31 Local removal of breast	1 year 3 months	N.S.D.
118	35	2 weeks	Massive. Inoperable	10 cm.	16.5.30 Ra. breast : 24 mgrm. L. areas : 23 mgrm. 168 hours	1 year 3 months	N.S.D.
119	52	2 years	Skin attached. Operable	3 cm.	17.4.30 Ra. breast : 63 mgrm. L. areas : 30 mgrm. 168 hours	1 year 1 month	Died Visceral metastases
120	53	5 months	Craggy mass, skin attached. Operable	6 x 4 cm.	18.9.30 Ra. breast : 33 mgrm. L. areas : 6 mgrm. 20.11.30 Ra. to other breast : 47 mgrm. 168 hours	1 year 3 months	Disease arrested
121	45	March, 1930, lump removed from breast		+	5.6.30 Ra. breast : 33 mgrm. L. areas : 20 mgrm. 168 hours	1 year 3 months	N.S.D.
				-	10.6.30 Ra. breast : 46 mgrm. L. areas : 28 mgrm. 168 hours	1 year 3 months	N.S.D.
				-	9.1.31 Local removal of breast	1 year 3 months	N.S.D.
				-	11.6.30 Ra. breast : 38 mgrm. L. areas : 23 mgrm. 168 hours	1 year 3 months	N.S.D.

## ANALYSIS OF 171 CASES OF CANCER OF BREAST TREATED BY RADIUM—continued.

No.	AGE	HISTORY	TYPE OF TUMOUR	MICRO-SCOPICAL SECTION	SIZE OF TUMOUR	GLANDS	TREATMENT	TIME	RESULT TO 13.3.31
122	57	3 months	Skin attached. Operable		6 × 4 in.	+	15.5.30 Ra. breast : 33 mgrm. L. areas : 26 mgrm. 168 hours	1 year 1 month	Died Thoracic and spinal metastases
123	61	1 year	Massive, skin infiltrated. Inoperable		6 cm.	+	19.6.30 Ra. breast : 59 mgrm. L. areas : 25 mgrm. 168 hours 26.3.31 Ra. breast : 39 mgrm. Axilla : 24 mgrm. 168 hours	1 year 2 months	N.S.D.
124	64	2 weeks	Adherent to skin and fascia, nipple retracted. Operable	A +	4 × 3.5 cm.	—	20.6.30 Ra. breast : 32 mgrm. L. areas : 32 mgrm. 168 hours 20.2.31 Excision of residual lump	1 year 2 months	N.S.D.
125	46	6 months	Massive, skin infiltrated. Operable		9 × 7 cm.	—	26.6.30 Ra. breast : 58 mgrm. L. areas : 26 mgrm. 168 hours	1 year 2 months	N.S.D.
126	57	1 year	Ulcerated, adherent. Inoperable		5 × 4 cm.	—	11.7.30 Ra. breast : 30 mgrm. L. areas : 26 mgrm. 168 hours	1 year 2 months	Disease arrested
127	52	3 months	Not adherent. Operable		4 cm.	+	17.7.30 Ra. breast : 46 mgrm. L. areas : 20 mgrm. 168 hours	1 year 1 month	N.S.D.
128	73	18 months	Ulcerated, skin infiltrated, not adherent. Operable	A +	7 cm.	—	17.7.30 Ra. breast : 34 mgrm. L. areas : 29 mgrm. 168 hours 11.8.30 Ra. breast : 30 mgrm. 168 hours 8.1.31 Excision of residual lump	1 year 1 month	N.S.D.
129	52	3 months	Not attached. Operable		5 cm.	+	26.8.30 Ra. breast : 40 mgrm. L. areas : 27 mgrm. 168 hours	6 months	Died Visceral meta- stases
130	61	15 months	Not adherent. Operable		3 cm.	+	28.8.30 Ra. breast : 21 mgrm. L. areas : 29 mgrm. 168 hours	1 year	N.S.D.

# RADIUM TREATMENT OF BREAST CANCER 475

131	46	3 weeks	10.7.30, Small lump excised from breast	B +	7 cm.	6.9.30 Ra. breast : 30 mgrm. L. areas : 20 mgrm. 216 hours	1 year	N.S.D.
132	75	1 year	Massive, ulcerated, skin infiltrated widely. Inoperable			17.9.30 Ra. breast : 58 mgrm. L. areas : 31 mgrm. 240 hours	11 months	N.S.D.
133	45	6 months	16.9.30, Excision of lump from breast	B +		3.10.30 Ra. breast : 43 mgrm. L. areas : 23 mgrm. 168 hours	11 months	N.S.D.
134	64	5 years	Contracting, adherent. Inoperable.		3 cm.	9.10.30 Ra. breast : 58 mgrm. L. areas : 21 mgrm. 168 hours	11 months	N.S.D.
135	49	2 months	Skin adherent. Operable		1.5 cm.	9.10.30 Ra. breast : 46 mgrm. L. areas : 26 mgrm. 168 hours	11 months	N.S.D.
136	45	18 months	Skin adherent, deviation of nipple. Operable		4 cm.	14.10.30 Ra. breast : 54 mgrm. L. areas : 32 mgrm. 168 hours	11 months	N.S.D.
137	41	4 months	Small, not adherent, Operable		3 cm.	24.10.30 Ra. breast : 45 mgrm. L. areas : 16 mgrm. 168 hours	10 months	N.S.D.
138	36	1 week	Skin adherent, nipple retracted. Operable		4 cm.	30.10.30 Ra. breast : 36 mgrm. L. areas : 29 mgrm. 168 hours	10 months	N.S.D.
139	58	About 4 months	Nipple retracted and adherent. Operable		3 x 2 cm.	31.10.30 Ra. breast : 46 mgrm. L. areas : 30 mgrm. 168 hours	10 months	N.S.D.
140	47	August, 1930, lump excised from breast	B +		+	31.10.30 Ra. breast : 45 mgrm. L. areas : 30 mgrm. 168 hours	7 months	Died Mediastinal metastases
141	64	4 months	Not attached. Operable	A -	3 cm.	28.11.30 Ra. breast : 52 mgrm. L. areas : 36 mgrm. 168 hours Excision of residual lump	9 months	N.S.D.
142	60	1 year	Skin dimpled, nipple retracted. Operable		3 cm.	28.11.30 Ra. breast : 35 mgrm. L. areas : 29 mgrm. 155 hours	9 months	N.S.D.

ANALYSIS OF 171 CASES OF CANCER OF BREAST TREATED BY RADIUM—continued.

No.	AGE	HISTORY	TYPE OF TUMOUR	MICRO-SCOPICAL SECTION	SIZE OF TUMOUR	GLANDS	TREATMENT	TIME	RESULT TO 15.9.31
143	72	2 years	Massive, fixed, skin infiltrated. Inoperable		10 × 9 cm.	+	27.11.30 Ra. breast : 48 mgrm. L. areas : 33 mgrm. 168 hours 25.6.31 Ra. residual nodule : 20 mgrm. 168 hours	9 months	N.S.D.
144	62	21.11.30, local removal of Rt. nipple and areola Lt. breast, small, attached to nipple. Operable		B +	3 cm.	—	3.12.30 Ra. Lt. breast : 46 mgrm. L. areas : 24 mgrm. 168 hours 10.12.30 Ra. Rt. breast : 44 mgrm. L. areas : 24 mgrm. 168 hours	9 months	N.S.D.
145	68	9 weeks	Skin dimpled. Operable		3 cm.	—	4.12.30 Ra. breast : 55 mgrm. L. areas : 29 mgrm. 168 hours	9 months	Improving
146	69	5 months	Small, skin dimpled. Operable		3 cm.	—	5.12.30 Ra. breast : 36 mgrm. L. areas : 20 mgrm. 159 hours	9 months	N.S.D.
147	53	1 year	Not adherent. Operable	A —	3 cm.	—	12.12.30 Ra. breast : 56 mgrm. L. areas : 21 mgrm. 216 hours 14.8.31 Excision of residual lump	9 months	N.S.D.
148	60	6½ years	Not attached. Operable		3 cm.	—	8.1.31 Ra. breast : 43 mgrm. L. areas : 26 mgrm. 168 hours	8 months	N.S.D.
149	64	2 years	Massive, fungating, ulcerated. Inoperable		8 cm.	—	19.1.31 Ra. Lt. breast : 52 mgrm. L. areas : 24 mgrm. 168 hours 16.6.31 Ra. Lt. breast : 31 mgrm. Rt. breast : 44 mgrm. 168 hours	7 months	Healing Retroclavicular extension
150	67	2 months	Small, not attached. Operable		3 cm.	+	26.1.31 Ra. breast : 40 mgrm. L. areas : 22 mgrm. 168 hours	7 months	N.S.D.
151	67	3 years	Contracting, skin attached. Operable		3 cm.	+	6.2.31 Ra. breast : 43 mgrm. L. areas : 32 mgrm. 168 hours	7 months	N.S.D.

# RADIUM TREATMENT OF BREAST CANCER 477

152	39	9 months	Small, skin attached. Operable		2.5 cm.	+	12.2.31	Ra. breast : 45 mgrm. L. areas : 23 mgrm. 168 hours	7 months	Improving
153	40	2 months	Not adherent. Operable		4 cm.	-	16.2.31	Ra. breast : 44 mgrm. L. areas : 30 mgrm. 168 hours	6 months	N.S.D.
154	55	6 years	Massive, skin infil- trated. Inoperable	B +	7 cm.	+	27.2.31	Ra. breast : 57 mgrm. L. areas : 26 mgrm. 168 hours	6 months	N.S.D.
155	54	1 month	Not adherent. Operable	B +	indefinite	-	23.1.31 6.3.31	Local removal of breast Ra. breast : 48 mgrm. L. areas : 28 mgrm. 168 hours	8 months	N.S.D.
156	44	2 months	Skin adherent, nipple retracted. Operable		4 cm.	+	9.3.31	Ra. breast : 57 mgrm. L. areas : 30 mgrm. 168 hours	6 months	N.S.D.
157	70	3 months	Contracting, skin adherent. Operable		4 cm.	+	2.4.31	Ra. breast : 39 mgrm. L. areas : 33 mgrm. 168 hours	5 months	Improving
158	62	8 months	Massive, skin adherent. Operable		7 cm.	-	2.4.31	Ra. breast : 70 mgrm. L. areas : 26 mgrm. 168 hours	5 months	Improving
159	59	5 months	Small, skin dimpled. Operable	B +	3 cm.	-	18.2.31 9.4.31	Local removal of lump Ra. breast : 48 mgrm. L. areas : 30 mgrm. 168 hours	5 months	N.S.D.
160	56	7 months	Small, skin adherent. Operable	B +	3 cm.	-	17.4.31	Local removal of lump and insertion of Ra. Ra. breast : 30 mgrm. L. areas : 42 mgrm. 168 hours	4 months	N.S.D.
161	61	18 months	Contracting, skin at- tached. Operable	B +	2.5 cm.	-	30.3.31 27.4.31	Local removal of breast Ra. breast : 21 mgrm. L. areas : 23 mgrm. 168 hours	5 months	N.S.D.
162	69	1 year	Massive. Inoperable		7 cm.	+	26.5.31	Ra. breast : 53 mgrm. L. areas : 36 mgrm. 168 hours	3 months	Improving
163	56	3 months	Massive, adherent, skin infiltrated. Inoperable		8 cm.	++	29.5.31	Ra. breast : 80 mgrm. L. areas : 22 mgrm. 168 hours	3 months	N.S.D.

## ANALYSIS OF 171 CASES OF CANCER OF BREAST TREATED BY RADIUM—continued.

No.	AGE	HISTORY	TYPE OF TUMOUR	MICRO-SCOPICAL SECTION	SIZE OF TUMOUR	GLANDS	TREATMENT	TIME	RESULT TO 15.9.31
164	63	3 months	Massive, not adherent. Operable		7 cm.	—	13.11.30 Ra. breast : 35 mgrm. L. areas : 29 mgrm. 168 hours	10 months	N.S.D.
165	50	1 month	Not adherent. Operable	B +	5 × 3 cm.	—	8.5.31 Local removal of breast 19.6.31 Ra. breast : 21 mgrm. L. areas : 31 mgrm. 168 hours	4 months	N.S.D.
166	56	5 months	Massive, skin attached. Operable	B +	6 cm.	+	29.5.31 Local removal of breast 26.6.31 Ra. breast : 24 mgrm. L. areas : 20 mgrm. 168 hours	3 months	N.S.D.
167	45	1 month	Skin dimpled. Operable	B +	3 cm.	—	8.6.31 Local removal of breast 2.7.31 Ra. breast : 12 mgrm. L. areas : 32 mgrm. 168 hours	3 months	N.S.D.
168	42	18 months	Massive, adherent, skin infiltrated. Inoperable		8 × 9 cm.	++	9.7.31 Ra. breast : 42 mgrm. L. areas : 41 mgrm. 168 hours	2 months	Improving
169	47	1 year	Contracting, adherent. Operable		4 cm.	—	9.7.31 Ra. breast : 63 mgrm. L. areas : 38 mgrm. 168 hours	2 months	Improving
170	41	6 weeks	Soft mobile lump. Operable	B +	3 × 2 cm.	—	23.6.31 Local removal of lump 10.7.31 Ra. breast : 24 mgrm. L. areas : 28 mgrm. 168 hours	2 months	N.S.D.
171	60	2 years	Skin adherent, nipple retracted. Operable		5 × 2.5 cm.	+	18.7.31 Ra. breast : 30 mgrm. L. areas : 27 mgrm. 168 hours	2 months	Improving

# RADIUM TREATMENT OF BREAST CANCER 479

## NOTES ON THE STATISTICAL ANALYSIS OF THE CASES.\*

By DR. JANET E. FORBER (*née* LANE-CLAYTON).

The most important group of cases is that in which the treatment was begun three or more years ago. There are 46 of these. In order to enable an ultimate comparison to be made with results obtained at three years by surgery alone, these 46 cases have been divided into three classes:—

Class I. Operable; no glands palpable.

Class II. Operable; glands palpable.

Class III. Inoperable.

As a matter of interest, results obtained with patients treated over two and under three years ago, and also over one year and under two years ago, have been taken out and are shown in the table below.

TOTAL CASES		ALIVE AT 3 YEARS	DIED OF CANCER	DIED OF INTERCURRENT DISEASE	PERCENTAGE ALIVE AT 3 YEARS
<i>Patients treated three or more years ago.</i>					
Class I	..	9	7	0	77.7
Class II	..	11	4	2	36.3
Class III	..	26	12	1	46.1
<i>Patients treated over two and under three years ago.</i>					
Class I	..	16	13	3	81.25
Class II	..	16	11	5	68.75
Class III	..	5	0	5	0.0
<i>Patients treated over one year and under two years ago.</i>					
Class I	..	22	20	2	90.9
Class II	..	16	10	5	62.5
Class III	..	10	5	5	50.0

Note.—Of those patients at three years, 3 in Class I, 1 in Class II, and 4 in Class III had either died of cancer or were dying at the time of writing. One of these in Class I died of a malignant ovarian tumour. Accepting the results published by the Ministry of Health (*loc. cit.*, p. 33) the percentage of those treated by surgery alone and alive at three years in Class I was between 75 and 78, and in Class II between 41 and 44.

Attention should be drawn to the fact that the classification of the operable cases is necessarily based on the palpation of the glands and not on the findings at the time of operation, or after microscopical examination. This renders it almost certain that some of the cases appearing in Class I had glands which were already invaded by growth, while Class II includes some very advanced, but still operable, cases. In comparing the results with those obtained by operation alone these sources of divergence are probably of considerable importance. Further, two cases, with no palpable glands but with ulceration of the skin, have been placed in Class II, since, with

\* Dr. J. E. Forber has very kindly compiled these notes from the proofs of the detailed table on pp. 460-478.



operation alone, these give very bad results, in no way comparable with Class I cases. (For an estimate of the errors cp. Chapters 6 and 7, *Ministry of Health Reports on Public Health and Medical Subjects*. No. 51.)

Two cases (one over three years since treatment and one over one year), both locally operable but having remote metastases at the time of treatment, have been classed among the inoperable ones, since it is unlikely that they would in fact have been operated upon had this been the only method of treatment available. Both have died.

The present sample is small, and does not enable any reliable data to be taken out to show the differences in results which there may be in the various methods of treatment, i.e., by radiology alone, or combined with surgery. A number of patients who died showed no local recurrence of the disease, but these have not been shown separately in the table.

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- <sup>2</sup> *Brit. Med. Jour.*, 1929, i, 242.
- <sup>3</sup> CADE, STANFORD, *Radium Treatment of Cancer*, 1929, 79. London.
- <sup>4</sup> CHEATLE, SIR LENTHAL, *Brit. Med. Jour.*, 1930, i, 807.
- <sup>5</sup> HANDLEY, W. SAMPSON, *Surg. Gynecol. and Obst.*, 1927, Dec., 721.
- <sup>6</sup> CUTLER, MAX, *Ibid.*, 1929, June, 217.
- <sup>7</sup> BIRKETT, G. E., *Radium Therapy*, 1931, 102. London.

## ON THE EFFECT OF ANÆMIA ON THE REACTIONS OF THE SKIN AND OF TUMOURS TO RADIUM EXPOSURE.

By J. C. MOTTRAM AND ALBERT EIDINOW,

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THE effect of radiation on the skin during control of the blood-supply was investigated by one of us.<sup>1</sup> The tail of the rat was used, and it was found that if the tail was blanched before radiation by winding rubber tubing up it, and kept blanched during the period of radiation by a ligature at the base, then the subsequent skin reaction was much less than when a normal tail was radiated. On the contrary, when the tail was made hyperæmic by being subjected to cold at 0° C., then the skin reaction following radiation was increased.

Jolly and Ferroux<sup>2</sup> obtained a similar result by tying the iliac artery, and studying the effects by X rays on the iliac gland: they found that tying the artery greatly diminished the effect of the X rays.

Why a diminished blood-flow results in a diminished sensitiveness, and vice versa, is unknown. It has, however, occurred to us that this fact may account for the small response to X-ray and radium treatment generally seen in anæmic patients; and the following experiments have been made to see whether rats rendered anæmic by bleeding show a diminished reaction to radium. Two sets of experiments have been carried out, one dealing with skin reactions, and the other with effects upon the growth of Jensen's rat sarcoma. The animals were bled by cardiac puncture, from 1 to 5 c.c. of blood being removed. Beta radiation was used from a square applicator of 60 mgrm., area 4 sq. cm., screen 0.12 mm. silver. Gamma radiation was used on six occasions, in which cases the applicator was screened with 3 mm. of lead in addition to the silver.

### SKIN REACTIONS.

When the skin on the clipped flank of a rat is given an exposure of one hour to the applicator already described, an erythema occurs on about the tenth day. Erythema is, however, an evasive sign, coming and going according to whether the rat is warm or cold. Desquamation of the epithelium and falling of the hair is the next occurrence, on about the thirteenth day. When the epithelium desquamates, a moist surface discharging serum usually results. If the reaction is not so severe, a crust of dried serum forms, and in any case the crust forms as soon as the moist surface begins to heal. Later the crust falls off, leaving a smooth glossy hairless skin, and finally the hair grows again.

Thus the formation of a moist surface (M.S.) and the falling out of the hair (H.O.) occur early, or late, or not at all, according to whether the reaction

is severe or mild. Afterwards the formation of a crust (Cr.), the falling of the crust (Cr.F.), and the re-growth of hair (H.G.), occur in this case late or early according to whether the reaction is severe or mild. The experiments consisted in exposing the flank on one side to the applicator, then bleeding the rat, and exposing the opposite flank to a similar dose of radiation. In the first set five rats were used; their weight, the amount of blood taken, and the minutes exposure to  $\beta$  radiation are given in *Table I*.

*Table I.*

RAT	WEIGHT	BLOOD TAKEN	RADIATION
	Grm.	c.c.	Mins.
1	250	1.5	60
2	100	1.75	60
3	180	2.0	50
4	95	2.0	60
5	250	2.5	60

The skin reactions are given in *Table II*, the figures representing the number of days after radiation that the skin reactions occurred; for instance, taking the case of the first rat on the control side (C), a moist surface (M.S.) occurred on the thirteenth day after radiation, the hair fell (H.O.) also on the thirteenth day, a crust (Cr.) formed on the seventeenth day and fell off (Cr. F.) on the twenty-second day, the hair re-grew (H.G.) on the thirty-fourth day.

*Table II.*

RAT	M.S.	H.O.	Cr.	Cr.F.	H.G.
1 E	—	15	—	—	28
1 C	13	13	17	22	34
2 E	—	23	—	—	36
2 C	20	20	28	36	42
3 E	13	13	15	?	36
3 C	13	13	17	?	36
4 E	—	28	—	—	31
4 C	20	20	23	28	39
5 E	16	16	20	23	31
5 C	13	14	20	36	38

In the case of the side radiated after bleeding (E) the reaction was much less, there was no moist surface or crust formation, the hair fell (H.O.) on the fifteenth day, later than on the control side, and the hair re-grew (H.G.) on the twenty-eighth day, much sooner than on the control side. The table shows that, with the exception of No. 3, where there is only a small difference, the skin reactions are decidedly diminished by bleeding the animal.

# EFFECT OF ANÆMIA ON RADIATION 483

A similar set of experiments was carried out with  $\gamma$  radiation using six rats. The details are given in *Tables III* and *IV*; in this case different rats were used as controls, since it was not possible to give a rat two doses of 15 hours' radiation.

*Table III.*

RAT	WEIGHT	BLOOD TAKEN	RADIATION
	Grm.	c.c.	Hours
1 E	145	2.2	16
2 C	145	No bleeding	16
3 E	270	4	15
4 C	285	No bleeding	15
5 E	245	5	15
6 C	250	No bleeding	15

*Table IV.*

RAT	M.S.	H.O.	CR.	CR.F.	H.G.
1 E	—	20	—	—	24
2 C	20	20	22	?	45
3 E	—	—	—	—	—
4 C	19	19	21	24	38
5 E	—	22	—	—	38
6 C	—	19	—	—	44

The tables show that the reaction of the skin to  $\gamma$  radiation is reduced by previous bleeding of the animal.

In the next set four were radiated on both flanks but without any intermediate bleeding, in order to make sure that radiation on one side did not effect the sensibility of the skin of the other, apart from bleeding the animals. The results, given in *Table V* show that there was no difference in sensitiveness.

*Table V.*

RAT	WEIGHT	RADIATION	M.S.	H.O.	CR.	CR.F.	H.G.
	Grm.	Mins.					
1 Right	60	60	—	15	—	—	30
1 Left		60	—	15	—	—	31
2 Right	250	50	—	18	21	24	33
2 Left		50	—	18	21	25	33
3 Right	90	60	13	13	15	30	?
3 Left		60	13	13	15	30	?
4 Right	100	60	—	16	15	19	29
4 Left		60	—	16	15	22	29

*Fig. 360* illustrates the results which are obtained. It shows the right and left sides of two rats which were radiated for one hour on the flanks, on the left side before bleeding, and on the right immediately after bleeding. On the left side ulcers are seen; on the right no skin reaction is visible.

The hair has been cut with seissors on both sides. The rats weighed 95 and 100 grm. ; 2 e.e. and 1.75 e.e. of blood were withdrawn from the heart. The photographs were taken twenty days after radiation.



FIG. 360.—Photographs of rats twenty days after radiation. (See text.)

TUMOUR REACTIONS

Rats were inoculated on both flanks with small pieces of Jensen’s rat sareoma. When the grafts had grown into measurable tumours, the larger of the two was given an exposure to  $\beta$  radiation, the animal was then bled, and then the other tumour was given a similar exposure. Subsequently the

Table VI.

RAT	WEIGHT	RADIATION	BLOOD TAKEN	TUMOUR
	Grm.	Mins.	c.c.	
1	55	35	1.5	Grew
2	110	35	1.0	Grew
3	75	35	2.0	Grew
4	?	45	0.8	Grew
5	?	45	1.0	Grew
6	?	35	1.0	Grew
7	85	20	3.0	Diss.
8	110	20	2.25	Diss.
9	115	30	1.2	Diss.
10	95	20	2.0	Diss.
11	110	35	1.75	Grew

tumours were measured at intervals of a few days and the superfieial area in square millimetres was obtained.

Eighteen rats were subjected to these experimental conditions, and in the ease of eleven animals there were no decided differences in tumour growth

on the two sides. *Table VI* gives the weight of the animals, the amount of radiation, and the number of cubic centimetres of blood withdrawn. In four cases both tumours rapidly disappeared, so that differences in growth-rate could not become manifest. In the other seven cases the tumours grew

*Table VII.*

RAT	WEIGHT	RADIATION	BLOOD TAKEN
	Grm.	Mins.	c.c.
1	95	20	1.75
2	90	20	2.0
3	95	20	3.0
4	90	20	2.6
5	110	35	1.75
6	?	35	1.25
7	?	25	1.0

at an equal rate; in no case did the tumours radiated after bleeding grow more slowly than the tumour radiated before bleeding.

In the case of seven animals which showed differences in growth-rate, charts are given, and the experimental conditions are set forth in *Table VII*.

The charts (*Figs. 361-367*) show the rate of growth of the tumours, superficial areas in square millimetres (ordinates) being plotted against days after radiation (abscissæ). Each chart gives the growth of the two tumours in the same rat, the full line of the tumour radiated before bleeding, and the dotted line of the tumour radiated after bleeding.

The charts show that in all cases the tumours radiated after bleeding when the animals are anæmic grow faster than the control. This disparity in growth-rate, though it occurs when comparison is made between tumours growing in different normal rats, is only seen as a great rarity in tumours growing on the two sides of the same rat. The differences, therefore, must be due to the bleeding of the animals, since this is the

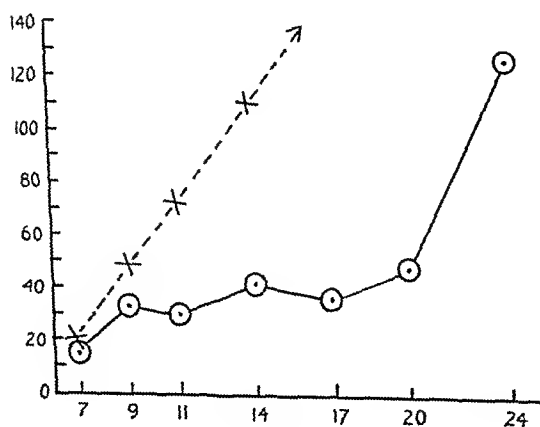


FIG. 361.

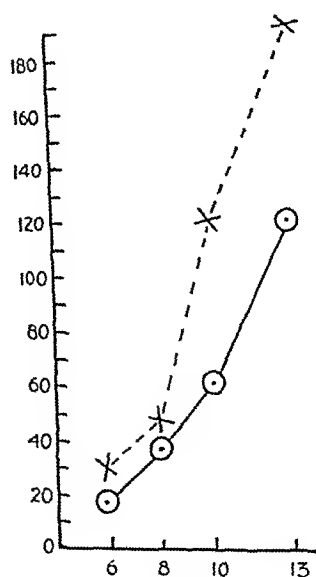


FIG. 362.

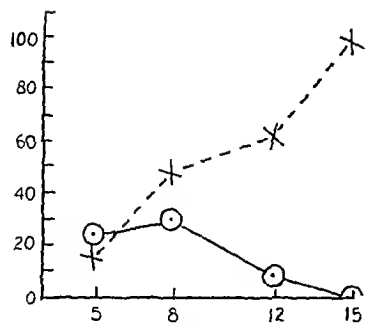


FIG. 363.

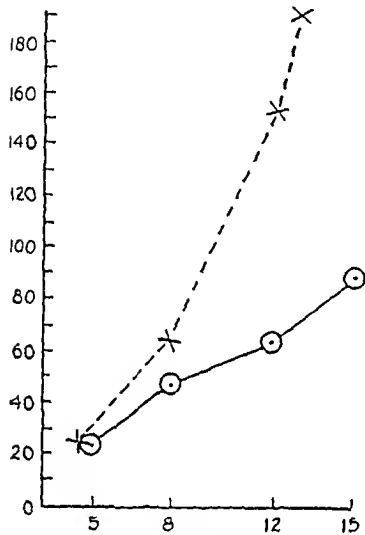


FIG. 364.

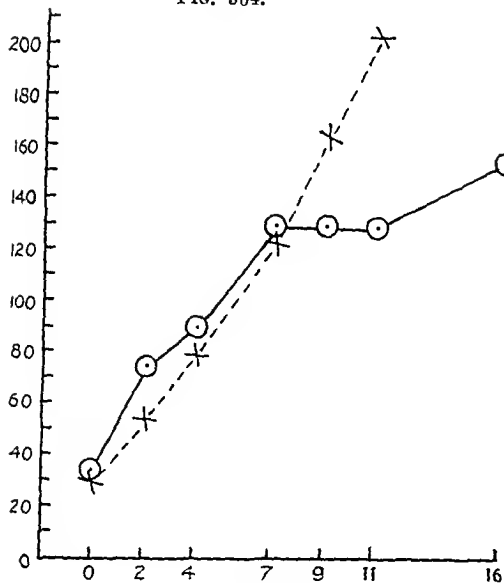


FIG. 366.

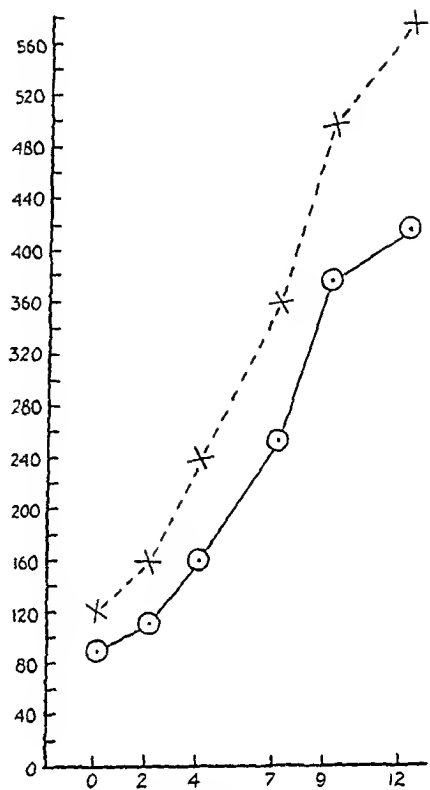


FIG. 365.

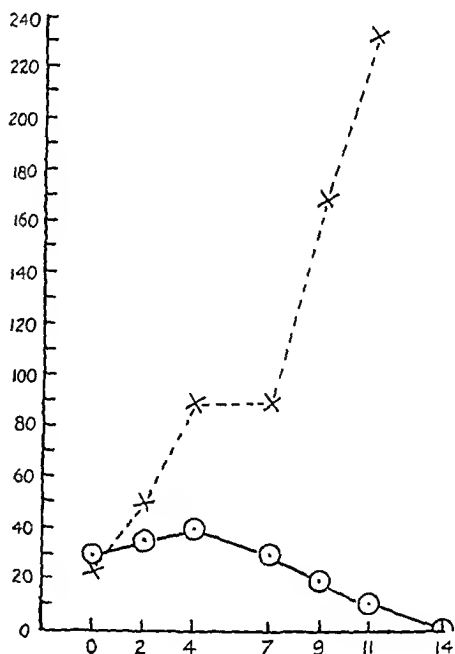


FIG. 367.

only variable experimental condition. In two animals, Nos. 4 and 8, not only was the growth-rate different, but whilst the tumours radiated before bleeding disappeared, that radiated after bleeding grew progressively.

The conclusion, therefore, is that tumours are rendered less sensitive to radiation by previous bleeding of the animal.

### DISCUSSION.

Why stoppage of the blood-flow during radiation renders tissues less sensitive to radiation is unknown. The explanation which seems to satisfy best all the facts with regard to the action of radiation on tissues is that the permeability of the endothelial cells is altered during radiation so as to allow some toxin substance present in the blood to enter and damage the endothelial cells. Bleeding would thus be effective in reducing reaction to radiation by diminishing the blood-flow to the tissues. Secondary radiation from some constituent of the blood, such as hæmoglobin, can be ruled out, since a diminished reaction occurs when the blood-flow is stopped though the blood-vessels are left in a state of congestion.<sup>1</sup>

Whatever the explanation, the clinical bearing of these experiments is clear and important, and they support the clinical experience that anæmic patients do not respond well to radiation, and that tumours with a poor blood-supply react badly to radiation. It follows that before treatment with radiation every endeavour should be made to bring the patient's blood to as near normal as possible. We are of opinion that blood transfusion should be thought of in this consideration. Further, when a series of treatments with radiation is being employed, any anæmia produced by radiation should be vigorously treated during the intervals between radiation, and the blood condition brought back to the normal before further treatment is given.

### CONCLUSIONS.

The bleeding of rats immediately before radiation renders the skin, and the tumour Jensen's rat sarcoma, less sensitive to  $\beta$  radiation than normal skin or tumours in unbled animals.

The radium used was on loan from the Medical Research Council.

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<sup>2</sup> JOLLY and FERROUX, *Comptes rend. Soc. de Biol.*, 1925, xcii, 67 and 125.



## THE PATHOLOGY AND TREATMENT OF TUBERCULOSIS OF THE KNEE-JOINT.\*

By G. R. GIRDLESTONE,

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### PATHOLOGY.

TUBERCULOSIS of the knee is always secondary to active tuberculosis elsewhere, and in children almost always arises from long-established infection of the lymphatic glands, and though this primary lesion may have been present for a long time, the occurrence of the metastatic focus in the knee is a proof that it is active and that through it tubercle bacilli are reaching the blood-stream. Lymphatic infection, often previously unsuspected, is the root of the disease, forming a lesion inaccessible, insidious, and in issue more dangerous than that of the knee. For active lymphatic tuberculosis means tuberculous bacillæmia, frequent, or perhaps almost constant if the laboratory experiences with animals can be taken as a guide. Doubtless the degree and frequency of the bacillæmia depends on the activity of the disease in the glands; so, too, does the spread of tuberculosis within the thorax by progressive infiltration of the lymph-ducts.

We have, then, beside the knee, this lesion, capable of insidious advance, and continually giving out showers of tubercle bacilli which constitute an ever-recurring threat to life. And these are no idle threats, for the later are end-results investigated the higher is the rate of mortality, until it passes 20 per cent.

We learn from our experience that tuberculosis of the knee is not itself a mortal disease; if death comes, it is due to the development of tuberculosis elsewhere; and operative treatment of the knee, however necessary, and however successful, is applied to the flower and not to the root of the problem. With the flower gone, it is easy to forget the root; for with the removal of the focus in the knee goes the only obvious reason for prolonged general treatment in the open air. Indeed, there is grave risk of earning the old thrust, "The operation was successful but the patient died!" Only prolonged general treatment, followed by good environment through the years

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\* When I was asked to open a discussion on this subject before the Association of Surgeons—a subject which is of very special interest to orthopædic surgeons—I felt quite unable to do so adequately without the help of my colleagues; accordingly I circulated a questionnaire and thus learnt the opinions of fifty-five of my colleagues in the British Isles, Canada, the United States of America, France, Italy, Holland, and Sweden. They gave me generous and most valuable assistance; some, indeed, took a great deal of trouble in looking up cases and getting together figures. I am most grateful, and feel that this paper is valuable in so far as it represents the present-day views of experts on a subject about which we have learnt much in the last few years.

of lymphatic disease and tuberculous bacillæmia, will give the patient safety during the time he needs to kill or to encapsule *all the tubercle bacilli in his body*.

The commencement of a focus in or near a joint may be due to tubercle bacilli settling in an area devitalized by a blow or settling in such large numbers that they can survive without any such preparatory devitalization. I think from the medico-legal point of view the time relationship of injury and the appearance of signs and symptoms is critical. Probably, however, the number of bacilli sown, and the suitability of the soil, influence the rate of the development of the lesion.

A few years ago I was the unwilling subject of a valuable experiment in which the time data are reliable. On March 2, while hurriedly extracting with a long gouge some carious bone from the depths of a hip with severe pyogenic infection, I embedded the gouge deeply in the pulp and periosteum of my left middle finger. The wound healed per primam, and, with the danger of sepsis over and that of tuberculosis unsuspected, I gave the finger some hard work during the next few weeks. The area of the wound was a little tender, but no more than one would expect. It was not till *ten weeks* after the implantation that there was sufficient swelling and discomfort to make me feel that a diagnostic excision of tissue was advisable. This was done and one or two tubercle bacilli were found. *Seven weeks* later the tuberculosis began to spread up the tendon-sheath, a gland in the neck developed, and amputation of the finger was necessary.

#### SITE OF INFECTION.

The infection of the knee-joint may be: (1) In the synovial membrane; (2) In one or more foci in the bone on either side of the epiphysial cartilage. And where the disease originates in the bone the joint will sometimes show signs of *irritation* before it is *infected*. The site and extent of infection are of clinical importance: and by a careful study of stereoscopic radiograms it is possible to distinguish three groups, and to outline for each group the appropriate modifications of treatment.

**Group I.—Extra-articular. With Osseous Foci irritating but not yet infecting the Joint.**—As Calvé puts it, "In contiguity but not in continuity with the joint". One's first feeling is that prompt elimination of the focus by an extra-articular route is indicated to save the joint from infection. But Calvé urges caution; he fears that there may be a communication from focus to joint not shown in the radiograms, and that an extra-articular operation may lead to tuberculous infiltration of the wound track from the focus, which in the other direction communicates with the joint, forming a tuberculous sinus with the danger of secondary pyogenic infection of the track and the joint.

Extra-articular operation on a focus truly extra-articular will save a joint, but on a focus which falsely appears extra-articular may lead to its ultimate infection with pyogenic organisms. The choice between conservative and operative treatment in these cases must depend upon the confidence with which one can distinguish the focus walled off by healthy bone from the focus with the wall already softened by tuberculous infiltration. I think

that with good stereoscopic radiograms it is generally possible to distinguish these conditions. But when doubt remains the decision must be made on the same lines as that regarding arthroscopy (*see below*), i.e., that operation is only safe after the age of 10 or so, for then in the event of the focus being found to open into the joint, excision can be carried out, and the disaster feared by Calvé avoided. (*Fig. 368.*)



FIG. 368. — Extra-articular focus in the femur involving epiphysis and metaphysis. When first seen the knee was painful, swollen, hot, and tender, and almost fixed by muscular spasm. Early radiograms were negative. Treatment, conservative, from Nov. 6, 1928, to April 27, 1931, when a collection of inspissated pus was excised. This was shut off from its origin, which was therefore not explored. Healing per primam. Programme: prolonged protection by weight-bearing caliper. The joint itself appears to have escaped permanent damage, and there is prospect of free movement ultimately.

**Group II.—Focal-articular. With Osseous Foci opening into the Joint and discharging Tubercle Bacilli into it.**—Such internal fistulae seldom if ever heal with a freely mobile joint. For movement will almost inevitably tear off the cap or seal and reinfect the joint. Fusion alone can give security; it remains to *choose the time*, and in young children to *await the age*, for operation.

The time will be chosen in regard to the age and the general and local condition, and no weight given to hopes of obtaining safe and permanent healing with good mobility. For these hopes have always been vague, and now the answers of the great majority of my colleagues agree that they are futile. I put the question: *Can you give me any evidence of the healing of an osseous tuberculous focus, which has opened into the knee, with retention of a freely mobile joint?* Of 40 who answer this question, 35 answer definitely "No", some adding, "I don't believe it ever occurs." Calvé says, "I do not know of any cases of an osseous focus open into the joint with restoration of normal mobility. The invasion of the joint is always followed by ulceration of cartilage, and then by definite loss of articular function."

It is true that five correspondents quote a total of 10 cases in 5 of which extra-articular operations have been done or sinuses apparently leading to extra-articular foci have appeared, and in the end free movement has been obtained. But these numbers are minute in relation to the thousands of cases covered. They represent very exceptional cases in young children. Fortunately, under the scheme of treatment I suggest, such

cases will not undergo premature fusion, but will have ample time to demonstrate the promise of mobility before they reach the age for fusion.

On the whole I may take Elmslie's answer as representing the views of the great majority; he says: "No. I should say that when a bony focus has opened into the knee the best result obtainable is an ankylosis." Such a conclusion appears almost inevitable in view of the morbid anatomy; for while an endosteal focus may become walled off and the caseogranuloma

eneapsuled, for this wall to form it is necessary that the focus be completely surrounded by living vascular material. When the focus is open to the joint on one side no such healing wall can form on that side. If the opening is *away from* the weight-bearing surface it can be capped by adhesions, and these will limit movement. Such an occurrence furnishes an example of the danger of limited movement, for the cap may be torn off by a sudden wrench. On the other hand, if the opening is *on* the weight-bearing surface it can only be capped by the opposite member of the joint. This form of natural healing is equally unsatisfactory, for it commonly results in an unsound fibrous ankylosis—fibrous rather than bony on account of the persistence of the articular cartilage in the face of tuberculous; and unsound because the strain falls on the caseo-granuloma.

All these considerations lead one to the conclusion that: *A tuberculous knee with bony foci communicating with the joint should be excised, and the arthrodesis should be performed at a time chosen in view of the age, local and general condition.* (Fig. 369.)

**Group III.—Non-focal.** Without Visible Osseous Foci, or if there are bone lesions they are small superficial foci round the edges of the articulation rather than fistulae opening into it, i.e., *the tuberculosis is radiologically synovial*, though perhaps 'pseudo-synovial'. This group may be termed 'non-focal'. My own figures and experiences in this group have convinced me that in young children purely conservative treatment is very well worth while and should be most carefully and continuously carried through.

Indeed, it appears that there is for them good hope of recovery with complete or almost complete movement (see Table I). To ascertain the views of others I put the question:—

*Are there any data or conclusions as to the occurrence of purely synovial tuberculosis of the knee, its histology, and its prognostic significance?*

That purely synovial tuberculosis exists has been demonstrated many times at operation. For while excising a knee and aiming at a complete synovectomy, a surgeon inspects completely the articular surfaces of femur and tibia. Now 38 out of 41 of my colleagues who answer this question



FIG. 369.—Unsound natural ankylosis in a man of 60. Skiagram showing pathological fracture (minor injury) in neighbourhood of old tuberculous disease which had begun fifty-five years before. Active recrudescence followed and amputation was necessitated. A remarkable example of persistent latent disease, and the failure of natural ankylosis.

agree as to the existence of purely synovial infection, and most add that the outlook is relatively favourable. Only three out of the 41 say either that synovial tuberculosis always goes on to osseous, or that minute osseous foci always exist round the synovial edges. The latter point is of no *practical* importance—it suffices us to know that when in young children no bone cavities can be seen throughout a series of radiograms, and when these cases are treated well enough and long enough, the majority will recover with full movement. Soutter, of Boston, with 9 synovial cases out of 45, reports 3 with full movement, 3 with fair, 3 with unsound ankylosis. Fraser reports that of his synovial cases 17 per cent had free movement, 56 per cent limited movement, 21 per cent some form of ankylosis, and that 6 per cent subsequently came to excision. I do not know to what extent these figures refer to end-results. For my own cases, *see Tables I, II.*

Now and then a case which appears hopeful under conservative treatment will ultimately come to excision. Nevertheless in the non-focal group one can peacefully carry on with conservative treatment till free movement is achieved or failing that excision becomes inevitable. For the time is not wasted and cannot be better spent.

*Table I.*—CHILDREN WITH 'NON-FOCAL' TUBERCULOSIS OF THE KNEE. TREATED AND WATCHED FOR 5 YEARS OR MORE.

(Wingfield Orthopædic Hospital.)

	No. OF CASES (1-4)	PERIOD SINCE SET FREE FROM ALL SPLINTAGE OR RESTRAINT
Result: Full movement .. ..	9	Years $2\frac{1}{2}$ , 4, $6\frac{1}{2}$ , $2\frac{1}{2}$ , 2, 3, $3\frac{1}{2}$ , $2\frac{3}{4}$ , $2\frac{1}{2}$
Result: Good movements (i.e., $180^\circ$ to above right angle) .. ..	2	Years $3\frac{1}{2}$ , $3\frac{1}{2}$
Under test .. ..	1	Free, promising, but time insufficient
Still under treatment .. ..	1	Promising
Excised, sound union .. ..	1	

*Table II.*—DETAILS OF THE 9 CASES WITH FULL MOVEMENTS SHOWN IN *Table I.*

	AGE	PLASTER SPICA	CALIPER AND GUARDING PLASTER	CALIPER ONLY	TOTAL PERIOD OF SPLINTAGE	PERIOD SINCE SET FREE
	Years	Months	Months	Months	Years	Years
1	9	7 + 5 = 12*	6 + 36 = 42*	28	$6\frac{3}{4}$	$2\frac{1}{2}$
2	2	20	13	—	$2\frac{3}{4}$	4
3	11	9	21	18	4	$6\frac{1}{2}$
4	$1\frac{1}{2}$	7	15	7	$2\frac{1}{2}$	$2\frac{1}{2}$
5	5	4 + 10 = 14*	5 + 9 = 14*	9	3	2
6	$1\frac{3}{4}$	36	4	27	$5\frac{1}{2}$	3
7	9	8 + 8 = 16*	4 + 8 = 12*	21	4	$3\frac{3}{4}$
8	12	12	9	4	2	$2\frac{1}{4}$
9	7	8	?†	?†	?†	$2\frac{1}{2}$

\* Two periods—second due to signs of commencing reactivity after remitting guarding plaster in each case.

† Owing to defective notes these time periods cannot be ascertained.

N.B.—All children with reasonably good homes become out-patients during the second period.

## DIAGNOSIS.

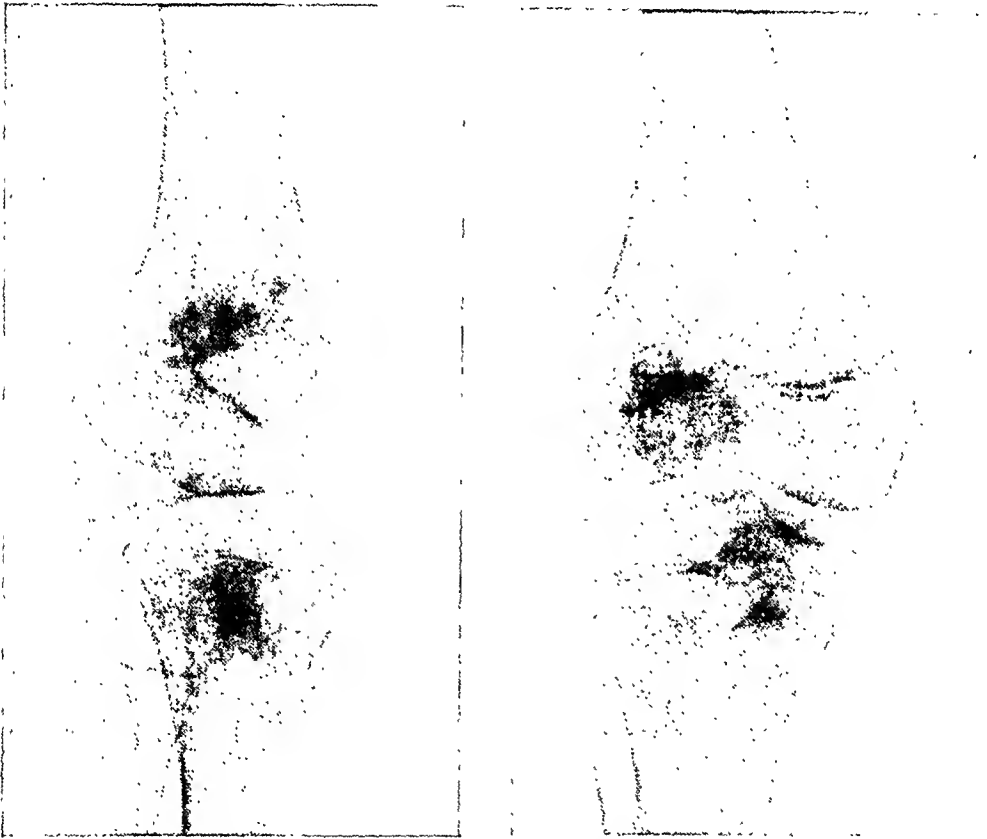
Now only prompt, effective, continuous, and prolonged treatment will prevent synovial disease from becoming osseous. Yet the nature of the disease is such that an early certain diagnosis is often impossible: therefore we must put up with a provisional diagnosis and act on it as quickly and stringently as if it were certain. Indeed, treatment and diagnosis must for a time run concurrently; so that it is impossible to avoid some consideration of diagnosis, though it is not included in the title of the discussion.

Tuberculosis of the knee is characteristically a monarticular arthritis with a very insidious onset. Generally the first thing noticed by the patient or parent is an interference with function and some swelling, rather than pain. Indeed, pain and tenderness are slight at first, the characteristic features being limitation of the full range of movement and a swollen, warm, and somewhat tender joint, with the enlargement due to swollen synovial membrane rather than to fluid. Inquiry may reveal: (1) The likelihood of human infection; (2) Previous manifestations of tuberculosis; (3) Some loss of health and vigour; (4) A history of an injury to the knee from six to twelve weeks previously, with a normal joint during most of the interval.

Both Calvé and Ombredanne mention enlargement of the regional glands in the groin, and, indeed, emphasize this as an aid in diagnosis. The muscular atrophy is often out of proportion to the history and the X-ray evidence. And while positive X-ray evidence is of very great value, negative by no means excludes tuberculosis. But in early cases it is impossible to make a diagnosis in the out-patient room. Therefore we must be content with a provisional diagnosis, for we cannot wait for the development of a conclusive clinical or radiographic picture. That is the very thing we want to avoid, and I feel this all the more keenly since I have learnt from my own end-results that in children under 10 without X-ray evidence of osseous foci early and very long-continued immobilization has led to a complete cure with full movements in the majority of cases. I think there had been a drift toward the feeling "they will all come to excision sooner or later". Fortunately we now know that this is untrue; but this knowledge adds to the urgency of our duty. Therefore I suggest that a provisional diagnosis of tuberculosis should be made on this slight clinical picture, whether radiograms reveal pathological changes or not; the knee should be immobilized at once and the patient admitted without delay for investigation.

On admission the patient is thoroughly examined, tonsils are removed if unhealthy, and the bowels regulated. Personally I have not found a blood-count helpful toward a diagnosis, either of tuberculosis itself, or of the cause of an arthritis simulating tuberculosis, and this I find is the general view. But I think that one should apply one or more tuberculin tests, preferably the intradermal (Mantoux), the Wassermann or sigma, and in some cases the gonococcal complement fixation test. The Wassermann or gonococcal tests have value when positive, though they do not, of course, eliminate the possibility of tuberculosis. (*Figs. 370, 371.*) Positive tuberculin tests have no value except perhaps in very young children, but negative tuberculin tests have great value. Let me quote Waldenström, of Stockholm, who says:

"In all cases I do the von Pirquet and Mantoux tests; if positive they give no help in the diagnosis; if negative, they tell us that the patient is free from tuberculosis—that is, also, in the knee." If, then, the tuberculin tests are positive and the other tests negative, we are no further on and must choose between a clinical diagnosis based on the march of clinical signs and radiograms and a diagnostic arthrotomy. The points on which such a choice can be made follow later. Of course if pus can be aspirated, a simple and decisive substitute for arthrotomy is offered and should be taken; but



FIGS. 370, 371.—Girl, aged 12. Syphilitic osteochondritis. Clinical condition resembled tuberculosis, and faulty diagnosis of tuberculosis was made. Subsequently Wassermann + + + +. Note relative density round epiphyseal disc of femur.

in my experience pus is very rarely to be found in early doubtful cases. Any clear or amber fluid there may give little or no useful information.

In the non-focal group, then, we are without radiographic help, and in young children for whom arthrotomy is inadvisable (*see later*) provisional diagnosis and immediate treatment is the only safe way of coming to a diagnosis; yet the early signs and symptoms are so slight and unalarming that it is hard to persuade oneself, and still harder to convince the patient and parents, of the necessity of his admission to hospital. One is tempted to hesitate, to try a month or two in plaster, and so on. This is a mistake.

Hesitation has led many a child down the broad and easy way that leads to destruction—of the knee-joint.

To hesitate is to be lost, for after a month or two of immobilization the knee appears almost normal. This disappearance of signs and symptoms is indeed the great trap in the diagnosis and treatment of tuberculosis of the knee. It is so tempting to treat the signs and symptoms and to remit treatment when they disappear. And synovial tuberculosis is only too ready to enter into this game of cat and mouse. Indeed, the disappearance of all signs of inflammation after a few weeks or months of immobilization should be regarded as part of the normal clinical picture of synovial tuberculosis.

Negative clinical and pathological information must be confirmed by the behaviour of the joint during test periods of gradually progressive lessening of the rest which has been enforced upon the joint. The patient should be *in hospital* or at least under good conditions and strict discipline during this test period. If the signs quickly disappear under immobilization—for example, have quite gone in six weeks—we remit strict immobilization but retain the patient in bed for Thomas's tests. If no sign of recurrence follows this freedom, further freedom is allowed, still under strict observation. And in a week or two more the child who has had an attack of transient arthritis from pharynx or intestine has set himself free and returned to normal life. On the other hand, any return of warmth, swelling, or limitation of movement indicates tuberculosis. All this applies to the child, not to the adult, to the synovial case, not to that with bone foci, and thus, it will be realized, to the one group of cases in which our care can be rewarded by a completely perfect recovery.

**The Value of a Clinical Diagnosis.**—Some years ago there was a reaction against too ready a diagnosis of tuberculosis of joints on insufficient grounds, and many demanded that the diagnosis should be restricted to those in which the radiographic evidence was very strong, or, better still, those in which tubercle bacilli could be demonstrated. This was a wholesome and corrective phase, but I have always felt that it was intrinsically unsound, so I asked my colleagues: *What evidence do you consider sufficient to make the diagnosis of tuberculosis?* Of 46 who answer this question, 39 are in general content with clinical diagnosis, but glad to have it confirmed whenever a chance of doing so without risk comes. Only 7 out of the 46 insist on a histological report or the finding of bacilli in fluid or tissues by the bacteriologist or the guinea-pig.

**Diagnostic Arthrotomy.**—Now, hitherto, the indications for a diagnostic arthrotomy have not been settled; hence my question: *Do you do a diagnostic arthrotomy? If so, what is your procedure?* That the indications had not yet been agreed on was clear from my answers: "Often or always", 15; "No or never", 15; "Rarely", "Occasionally", "Only in selected cases", 23. But I think my collaborators have enabled me to answer the question and to define clearly the indications and contra-indications. To begin with, it should be understood that diagnostic arthrotomy is not without danger both to the patient and to the diagnosis.

First as to the diagnosis—false negatives are common, for the histologist, bacteriologist, or guinea-pig returns a negative if the material supplied is



*inflamed* but not *infected* by the tubercle bacilli. For there is no characteristic histology *unless tubercle bacilli are present in the microscopic field* (the bacilli need not be *demonstrated*, for that is technically much more difficult than the recognition of the histology characteristic of their presence). This is analogous to the radiological position, for there is no characteristic radiographic picture unless the tubercle bacilli have been working in a radiographic field—that is, unless there are foci in the bone. And purely negative reports from the histologist, bacteriologist, and guinea-pig ought to be classified with negative reports from the radiologist.

Secondly as to the danger to the patient—the following are quotations from the reports of three of my collaborators.

First there is Osgood, of Boston, who writes that he does a diagnostic arthrotomy “only in very infrequent cases. Have been troubled by the occurrence of sinuses along the tracts of incision, appearing often several months after complete healing of wound. These unfortunate events have occurred chiefly in biopsies of the hip-joint. In early cases of suspected knee-joint tuberculous, danger exists, and material excised may be indeterminate unless thorough exploration of the joint is made.”

Then Ober, also of Boston, who has probably seen some of Osgood's cases, says: “I do not do a diagnostic arthrotomy except when I feel sure that it is not tuberculosis. At the New England Peabody Home we have had some very bad results as a result of a biopsy, tubercle bacillus invading the whole area of the wound, including the skin and scar about the wound, and finally breaking down; and these biopsies were done by those who have strongly advocated biopsy. Personally I consider the result of the biopsy very detrimental to the child. One ought to be able to diagnose syphilis and osteomyelitis from other elements and without biopsy. Arthritis is probably more common in children than we have suspected, so that I would say a biopsy would be done in the presence of a negative tuberculin reaction, not in the presence of a positive one.”

Thirdly there is Soutter. He rarely does arthrotomy, saying: “I have followed the method of arthrotomy for diagnosis of tuberculosis, and the men that have been doing it largely here are not doing it to any great extent. It should not be done in tuberculosis of the joint without good reason, and then only when the disease has already involved the joint.”

It is clear that the danger to the patient is tuberculous infiltration of the wound track outwards, followed by pyogenic infection inwards. The former is a slow affair and can always be prevented by arthrodesis within a week or two of the diagnostic arthrotomy. It must be true to say that diagnostic arthrotomy is *free from risk only if a positive diagnosis of tuberculosis will be quickly followed by fusion, and dangerous only when conservative treatment will be continued despite a positive diagnosis.*

I suggest that in young children a clinical diagnosis, carefully tested and reviewed, is all that one needs; arthrotomy is contra-indicated, for it is risky and unnecessary. The persistence of a chronic decalcifying arthritis, with or without bony foci, is sufficient for all practical purposes.

INDICATIONS FOR DIAGNOSTIC ARTHROTOMY.—Diagnostic arthrotomy is indicated:—

1. In young children—*never*, except perhaps when repeated tuberculin tests have excluded tuberculosis and some other cause is sought; this is Ober's opinion. Incidentally it may be mentioned that an arthrotomy is not the best way of distinguishing a hæmophilic knee. One of my collaborators mentions with pious gratitude that he was only prevented from this by noticing a bruise.

2. In cases radiologically non-ossæous—only when the patient is ripe in stage and in age for fusion.

3. In adults—always, as the first step of an excision.

When an arthrotomy is done, attention should be paid to the following points: (1) Ample incision into the joint for inspection of synovia and articular surfaces; or, if the signs are localized, a smaller incision in that region. (2) Discovery of the most diseased part and removal of a generous sample of this for histological examination. (3) It is well to ask the pathologist also to be generous in his supply of material to each guinea-pig, for the infection may be slight and scattered. Fraser suggests that two guinea-pigs are better than one. I agree, but I would rather give one guinea-pig a big dose than let two split it between them.

*Note.*—If for some extraordinary reason an arthrotomy is to be done on a case which may prove tuberculous, and which, if it does prove tuberculous, will be treated by conservative means, the edges of the capsule should be accurately protected before the synovia is opened, and very accurately sutured afterwards.

It is true that the ardent arthrotomist has served a very useful purpose by proving that knee-joints certainly infected with tuberculosis may run their course without radiographic evidence of any destructive lesion, and ultimately recover with full movement. This we now know, and for the future we can confine arthrotomy to those cases in which it will be helpful and harmless.

In my questionnaire I asked for opinions as to the results of the bacteriologists' reports on histology, on the finding of tubercle bacilli in tissues or pus, and guinea-pig inoculation. Now in Oxford we are very fortunate in having in Dr. A. D. Gardner a bacteriologist who has made a special study of this subject. He has given me the following table dealing with examination of synovia removed at arthrotomy in 18 cases which proved tuberculous.

Table III.

Positive Histologically	..	..	..	..	17 (94 per cent)
(One case was negative histologically and by guinea-pig, but proved by finding T.B. in smear)					
Positive by Guinea-pig Test	..	..	..	..	13 (72 per cent)
Positive histologically	{	..	..	..	4 (22 per cent)
Negative by guinea-pig					
Negative histologically					
Positive by guinea-pig	}	..	..	..	0

N.B.—For histology, from one to four selected blocks of tissue were prepared, according to whether the tissue looked obviously tuberculous or doubtful.

On the whole, the opinions of my colleagues put the guinea-pig above the pathologist, but Dr. Gardner's experiences show that a pathologist

experienced in tuberculous material, who is prepared to take time and trouble, is at least as good as a guinea-pig in finding the bacilli in pus and furnishing a diagnosis from tissue. For a portion of tissue sufficiently infected to give a guinea-pig tuberculosis will be histologically characteristic of tuberculosis.

Almost all my correspondents report false negatives. Those who want to pursue this subject further should read Ghormley's<sup>1</sup> interesting paper.

## TREATMENT.

### GENERAL TREATMENT.

As the patients suffer from tuberculosis, of which the knee is only one and the less dangerous lesion, they all need general treatment by physical rest and metabolic stimulation, heliotherapy, a well-chosen and varied diet, and enlivening environment.

In adults this general treatment is *preparatory to operation*; but it is wise to explain to the patient that the operation will not eliminate the need for general treatment afterwards. The infection of the knee is a proof of tuberculous bacillæmia, and another metastatic lesion is likely unless the source of the bacilli is dried up and the patient's resistance raised by general treatment. Good treatment will hasten, but nothing can cut short, the process of healing. Indeed, one wonders whether after early middle age the glandular disease ever heals quite soundly. Second lesions are unhappily common.

### LOCAL CONSERVATIVE TREATMENT.

The local conservative treatment consists in keeping the joint quiet and cool. Any heat or disturbance, e.g., diathermy or massage, applied to the joint is extremely harmful. As to the methods of immobilization, the great majority agree that so long as the joint is warm it should be completely immobilized, and that the best method for this period is a plaster spica including pelvis and foot. For example, Putti advises "rigorous immobilization in plaster for a long time".

Deformity due to soft tissue changes can be corrected gradually and easily as the inflammation dies down. Fixed deformity can be left until the fusion operation, and it is very possible, as MacLennan believes, that the healing is quicker with the knee somewhat flexed.

Some weeks or months after the knee has become cool a caliper with a guarding plaster can be applied and the child allowed up. Later the guarding plaster can be left off, but the caliper must remain for years. I am sure that it is true that no harm follows wearing a caliper unnecessarily long. Where the disease is synovial (or pseudo-synovial) free movement often follows long-continued and continuous immobilization. Immobilization for years does not lead to fixation, whereas lack of immobilization or a break in its continuity is liable to lead to extension and reactivity of the disease, and to destruction of the articular cartilage on whose integrity joint function depends. *Permanent loss of movement is the result of disease, or the operation it necessitates, never of immobilization.* A study of several series

of radiograms of non-focal knees has convinced me that the articular ends of femur and tibia usually develop normally during the years that they are immobilized. Free and normal movement returns of itself gradually and progressively if the knee is set free only after the disease is soundly healed.

The secret of the gratifying results which are being obtained by conservative treatment in children with synovial tuberculosis of the knee is threefold: (1) The effectiveness of immobilization; (2) The continuity of immobilization so that there is no re-arousal of the tuberculous inflammation; and (3) The length of immobilization.

The 'non-focal' knee should be kept in a caliper at least a year, probably two, after all signs and symptoms of activity (such as warmth, swelling, and tenderness) have disappeared, if it is to be completely and permanently safe. And in those with bony foci communicating with the joint there is no advantage in early excision (and there are some dangers). For the knee goes on quite happily in a caliper. And the longer the wait, the readier are the bones for fusion, as a result of recalcification, and fuller ossification of the epiphyses. The age at which fusion will be done in any particular case depends partly on the age of onset and subsequent length of efficient treatment, and on the recalcification of the bones as shown in radiograms.

In adults and adolescents, on the other hand, there is no advantage in continuing *local* conservative treatment once the activity of the disease is over. Radical local treatment is then followed by continued general treatment.

In short, the local disease, with varying distribution (synovial or bony) and varying activity, demands varying methods of immobilization; but the general condition is the main factor which determines the length of hospitalization, and of after-care.

#### OPERATIVE TREATMENT.

##### Excision, Fusion, Arthrodesis.—

1. There are only two safe end-results for a tuberculous knee—free movement or bony ankylosis.

2. Free movement is not obtainable in adults, and in children only to be sought when there is no radiographic evidence of osseous foci communicating with the joint.

3. In adults, then, ankylosis is the aim, but without operation ankylosis is delayed, very unlikely, and, if it occurs, dangerously unsound.

If these are true axioms, fusion is indicated in all cases of proved tuberculosis of the knee-joint, except in children without radiographic foci communicating with the joint, and cases in which it is contra-indicated by age or complications.

It is true that Rollier says that excision is never necessary. This extreme advocacy of conservative treatment is unsupported by any of my colleagues.

It remains to define the operation, to consider when and how it should be done, and what are the dangers and contra-indications. The modern operation, whether it is called excision, fusion, or arthrodesis, is designed: (1) To promote bony ankylosis; and (2) To remove all diseased parts which are accessible without either interfering with growth or lessening the prospect

of bony ankylosis. The dangers of the operation are listed later. In the main they are due to operating on cases *unduly early, unduly young, or unduly septic*.

#### INDICATIONS FOR EXCISION.—

1. *Age*.—All adolescents over 15 and adults under 50 or 60. Children who have osseous foci communicating with the joint, when they attain 'excision age'.

2. *Stage*.—(a) In *adolescents and adults*. Only after the general illness and local activity have been checked by general treatment and immobilization. (Incidentally during this period the various diagnostic tests can be carried through.) (b) In *children with osseous foci*. Only when conservative treatment is complete and the patient is old enough (? 10, 12, 14, 16—opinions differ). (c) In *children without osseous foci*. Only when the patient passes puberty without the promise of free movement.

3. *Confirmation of Diagnosis*.—After the diagnosis has been confirmed by arthrotomy which, if the naked eye suffices or frozen sections are available, can be the first step of the excision, or, if microscopic evidence is needed, can precede excision by a week or two.

CONTRA-INDICATIONS TO EXCISION.—These are: (1) Childhood; (2) Unchecked activity of the disease, general or local; (3) Active phthisis; (4) Extensive involvement of the femoral shaft; (5) Active septic infection of sinuses, despite preparatory drainage or free exposure, scraping, and packing.

In answer to my question whether a sinus should be looked upon as a contra-indication to arthrodesis 22 surgeons say "No", and express no fears; 14 qualify their negatives, indicating that they do consider it dangerous to excise if the infection is active—for example, Osgood says, "If infected danger must be appreciated", and Calvé says, "I do not consider the presence of a sinus a contra-indication to resection if not accompanied by febrile symptoms, and the knee as a whole is dry. In febrile cases or bad general condition—wide resection of tracks with removal of sequestra—never *resection* (of the joint)."

An American surgeon considers an infected sinus a contra-indication, saying, "My experience shows that it is very dangerous." An Englishman, "Yes, two cases, both did very badly." At the Wingfield Hospital only one excision has been done in the presence of an infected sinus. The patient, a middle-aged woman, died of pyæmia. Theoretical considerations and the experience of this case make me shrink from excision in the presence of a sinus.

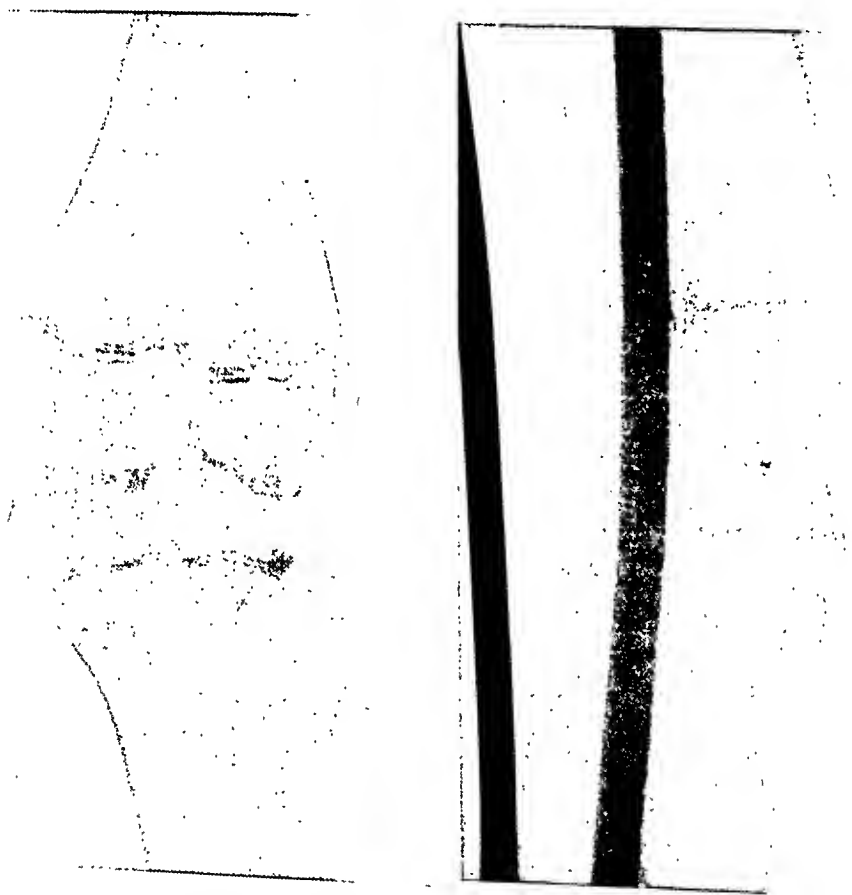
I feel that Calvé's method of preliminary laying open and resection of the sinus tracks and perhaps paraffin flavine dressings for a time is wise. I am in favour of very thorough drainage and continued immobilization. If in spite of this the sepsis and the tuberculosis remain persistently active, I would amputate rather than excise, for I must admit to being altogether averse from laying open in the presence of sepsis all the raw absorbent surfaces of bone and tissue entailed by excision and synovectomy.

#### TECHNICAL POINTS.—

1. *Tourniquet and Towelling*.—I think a tourniquet is valuable for the earlier stages of the operation, but it should be removed for *hæmostasis* before any sutures, grafts, pegs, or nails are applied. The tourniquet makes the

operation easier for the surgeon, and the patient loses less blood and must surely suffer less from toxæmia and bacillæmia. The towelling should be so folded and clipped round the limb above and below the knee that the leg is stockinged and the thigh trousered; otherwise gaps appear when the knee is fully flexed.

2. *Synovectomy*.—In answer to the question whether we should aim at complete synovectomy, 31 surgeons answered "Yes", 13 said "No", and 2 answered "In adults only". Of the 13 "Noes", most aim at removal of *all*



FIGS. 372, 373.—Boy aged 13. Illustrating minimal removal of bone.

*grossly diseased material*. Personally I always aim at a complete synovectomy in adults (and this I believe to be an essential safeguard) and at any age if the synovia appears diseased. In children, when the operation comes—properly—at the end of conservative treatment, fusion is needed, synovectomy not so; and I doubt whether there is any advantage in removing the suprapatellar pouch in these cases.

3. *Division of all Ligaments*.—After removal of the suprapatellar pouch, complete division of the mesial, lateral, and crucial ligaments facilitates the posterior synovectomy.

4. *Osseous Foci*.—If there are osseous foci, I scrape them out with a

sharp spoon. There is no need to level the bone down to their bases, and there are great disadvantages in doing so.

5. *Preparation of Bone Surfaces.*—Most of my colleagues use a broad saw and cut plane surfaces. Albee uses a narrow-bladed saw and shaves the femoral condyles, then cuts a coronal trough across the tibial tuberosities to fit. For adults this is excellent, but for children I prefer to use gouges and to shape each condyle to a ball and each tuberosity of the tibia to a shallow socket, leaving the bared tibial spines as a tongue fitting into the bared intercondylar notch. There is less removal of bone and the apposition is more secure (*Figs. 372, 373*).

6. *Angle.*—I am surprised to note that a few surgeons still advocate a straight knee. Most, I think, choose the angle to suit the sex and occupation of the patient. Probably an angle of about  $150^\circ$  is a good average for an adult. But the younger the patient, below 18, the less the angle that can safely be allowed on account of the strain imposed upon the tibial epiphysis by ankylosis in flexion.

7. *Removal of Tourniquet.*—The tourniquet should be removed as soon as synovectomy has been completed and the bone-ends have been prepared. All bleeding points are then tied.

8. *Internal Fixation.*—Most surgeons who cut the bone-ends square use some form of internal fixation. The favourite methods are decussating bone-pegs or long decussating nails which project from the plaster and are removed several weeks later. Some use the bared patella, but if one removes very little bone it must be used as a free graft. Tibial and hinged osteoperiosteal flaps are mentioned. But with the ball-and-socket method apposition is easily maintained, and alteration of the angle of flexion does not interfere with the area of bony contact: so that there is little need for internal fixation.

9. *Drainage.*—Drainage for twenty-four hours is always advisable.

10. *Method of Immobilization.*—A plaster spica including pelvis and foot is almost universally used. Flying buttresses of plaster or ferro-plaster enable one to cut a large window at once for dressing the wound and removing the blood drains.

11. *Length of Immobilization.*—It seems wise to leave the patient in the spica for ten or twelve weeks, then use a guarding plaster with a caliper flexed to fit until X-rays show sound union. (*Fig. 374*.)

#### THE RESULTS OF EXCISION.—

*Failure* may be due to:—

1. Operating while the disease is active, the patient ill, and the local resistance so poor that local tuberculous recrudescence takes place.

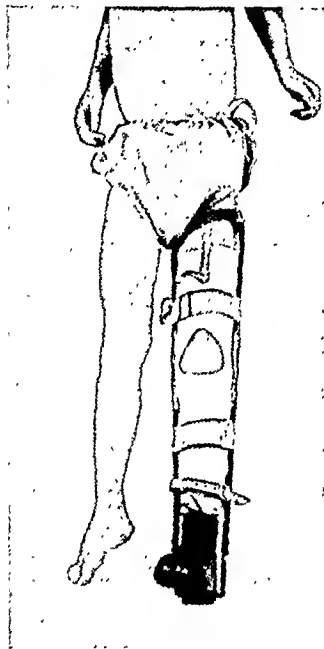


FIG. 374.—Caliper and guarding plaster. Note suspensory strap from caliper ring to buckle fixed in plaster.

2. *Active tuberculosis elsewhere*, making the patient too ill to produce union despite the absence of local tuberculous recrudescence. (One such case

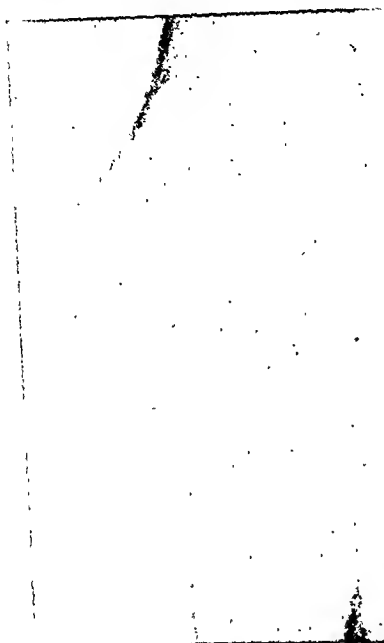


FIG. 375.—Tuberculous knee after excision. Patient had serious phthisis, and despite accurate fit and immobilization union did not take place.



FIG. 376.—Result of old-fashioned excision on young child. Failure in fusion and disastrous damage to epiphysal discs. (See Fig. 377.)

occurred at the Wingfield Hospital, the patient ultimately succumbing to phthisis a year or more after the operation—Fig. 375.)

3. *Pyogenic infection from a sinus*. I think there is considerable danger from this.

4. *Ineffective immobilization*, allowing displacement between the end of the operation and the completion of the plaster, or harmful mobility afterwards.

5. Operation done on a very young patient is liable to fail either from: (a) lack of bony union because the ossific nuclei in the epiphyses are relatively small, (b) because secondary deformity develops from the unusual strains thrown on the young weak bone laid down on the metaphysial side of the growth disc, or (c) the classic error of the past—namely, interference with growth from too radical operation resulting in damage to one or both discs. I have a girl of 20 under my care; she was

operated on elsewhere, and one of her legs is 7 in. short as a result of this catastrophe. (Figs. 376, 377.)



FIG. 377.—Photograph of case seen in Fig. 376, showing the amount of shortening.



*Success.*—I asked my colleagues for their experiences as to the percentage of bony ankylosis after excision. A few have returned actual figures, notably Henderson, of the Mayo Clinic, who reports 194 excisions, with bony ankylosis in 171 cases (88 per cent). A distinguished Italian surgeon records 104 cases with 66.6 per cent of bony ankylosis; 26 other correspondents have returned figures (mostly estimated), and these average 87 per cent. My own series done by my colleagues and myself covers 26 cases done long enough to furnish end-results, with 88 per cent of sound bony ankylosis. There were three failures; one was the middle-aged woman ill with a superadded pyogenic infection already mentioned; the second was a middle-aged man with phthisis who died from that condition between one and two years afterwards (*Fig. 375*); the third, an adolescent, whose joint had been very extensively destroyed, has a sound fibrous but not bony ankylosis; he has been hard at work for more than three years, and there is no clinical or radiographic sign of tuberculosis. In the remaining 23 cases sound bony ankylosis resulted. Of our three failures one is a relative success, and in the other two cases I now feel that excision was contra-indicated.

I do not know of any case in which excision has led to tuberculous dissemination. Doubtless that danger is minimized by the previous conservative treatment and the tourniquet.

Altogether, I think that *excision of the knee* comes out of the inquiry with an enhanced reputation. Yet since it leads to the loss of movement in a joint in which movement is very valuable, I suggest that it should be confined strictly to those cases in which we know from the type of the disease, or from the age of the patient, or after a prolonged trial of conservative measures, that it is futile to hope for movement with sound safe healing. But although a soundly ankylosed knee compares poorly with a normal joint, it is far better than a thigh stump. Excision is a safe second string to our bow; for it provides a sure means of eradicating the local disease in all but the senile, the septic, or the advanced consumptive. In all the others we can say, "We'll try to save the joint, but, if that fails, we can and *will* save the limb."

*Amputation.*—To my question, "*What do you consider the indications in the age groups 0-14, 15-50, and 50 upwards, for amputation?*", Elmslie's answer is representative. "Amputation under 14 only when the case does very badly with conservative treatment. Young adults only when doing badly under conservative treatment and if the bone disease is too extensive for excision. Older people in any case unsuitable for excision."

*Amputation is indicated only when either the patient or the limb has otherwise little prospect of recovery.* That means that amputation is indicated *very rarely*. It should be confined to the patient who is very old or very ill, or to the knee which is very extensively diseased or heavily infected with pyogenic organisms. (*See Table IV.*)

If ordinary anæsthesia is contra-indicated we can rely on spinal anæsthesia, or avertin with the mild complemental respiratory anæsthesia necessary.

*In children amputation should be left quite out of ordinary consideration; indeed, the number of our amputations in patients below middle age is a fair*

indication of our skill in the happier measures. In the last twelve years I have done one such amputation, and I think I ought to have done a second. but Waldenström can say that in all his twenty years at the S. Gorin Hospital he has only had to do a single amputation!

*Table IV.*—AMPUTATIONS FOR TUBERCULOSIS OF THE KNEE.  
(Wingfield Orthopædic Hospital.)

	AGE	REASON	RESULT
1	26	Very extensive pyogenic infection of fixed flexed knee	Good for a year or more, but subsequently developed lesions in spine and rib; still under treatment
2	39	Active phthisis	Good
3	60	Age*	Good, except for pain of phantom limb type
4	57	Age†	Good
5	36	Extensive pyogenic infection	Good
6	2	Active extending secondarily infected disease and general illness threatening life	Good
7	44	Active phthisis	Good

\* Recrudescence of disease after 52 years.

† Tubercle not diagnosed till pathological report following synovectomy.

Sir Robert Jones has taught us that in a patient past middle age tuberculosis must be dealt with almost as radically as malignant disease. It is the real age, the senility of the patient in relation to the extent and activity of the local disease, that counts. There are very few indeed whom Osler would have found 'too old at 40' for excision, and some perhaps who are too young at 60 for amputation. I think Freiberg conveys a useful hint when he says: "We feel that amputations are probably not done often enough in patients beyond middle age." But I should not be fair to my collaborators if I did not express their unanimous revulsion against amputation. It is, indeed, a last resort.

**Sinus Formation.**—Just one word about sinuses. There is a vast difference between a tuberculous sinus and a sinus secondarily infected. A tuberculous sinus will, as a rule, close quickly if the general and local treatment is good.

It is a danger because it exposes the knee to the risk of pyogenic infection; and this catastrophe can only be avoided by *very* careful antiseptic dressings. The skin must not become sodden or eezematous: we wipe from the sinus spirally onwards with weak iodine or biniodide in spirit, and fix the dressing securely so that it cannot wander. Above all a tuberculous sinus should *never be probed*.

**Arthroplasty.**—I asked my colleagues: *Has arthroplasty any place in the after-treatment of tuberculosis of the knee?* There were 44 answers to this question, and 32 "Noes". Fairbank underlined his "No" four times, and Calvé said "Criminal". Very convincing, too, is a plain "No" from Putti, probably the greatest exponent of arthroplasty of the knee. It is also

interesting to note that 15 out of the 19 American orthopædic surgeons who answered the question said "No".

There remain 11 who give a qualified consent to the idea that an arthroplasty might possibly be warranted under exceptional circumstances—for example, as Willis Campbell points out, when first one knee has had to be fused and later the other has suffered in the same way. The particular circumstances of the case might warrant an arthroplasty of the first knee provided the time interval and radiographic appearances justified it. Incidentally I note that Willis Campbell reports 57 arthroplasties, but not one for tuberculosis.<sup>2</sup>

Bankart points out that by the time the operation is bacteriologically safe it is generally an unsound practical proposition on account of the condition of the muscles controlling the joint. Indeed, a sound ankylosis of the knee in good position is likely to be better than the sort of arthroplasty that will result at a very late stage, after a very chronic disease; and the ankylosis is certainly better for a working man or woman.

The fibrous ankylosis following conservative treatment is seldom permanently safe from reerudescence, and of course still less safe for arthroplasty. Time alone does not bring safety. I have a case in which tuberculous reerudescence occurred fifty-two years after the original attack.

I would sum up the matter by saying that in cases resulting in *fibrous ankylosis*, *arthroplasty is unsafe*, and *after bony fusion the parts are unsuitable*. And I unreservedly join those who hold that there is no place for arthroplasty in tuberculosis of the knee.

### SUMMARY.

**Pathology.**—Tuberculosis of the knee is a dual disease—an outspoken lesion in the knee arising from another deep, unseen, and more dangerous lesion.

Cases can be classified into three groups by the topography of infection, and again into three groups by the age of the patient, and each group calls for a different line of treatment.

*A.—Three Groups Distinguishable by Stereoscopic Radiograms:—*

1. '*Extra-articular.*'—With purely extra-articular foci. The question of radical elimination of these foci by an extra-articular route is discussed, and Calvé's warning considered.

2. '*Focal-articular.*'—With bone foci communicating with the joint. Reasons are given for the decision that the joint should be fused, and the arthrodesis performed at a time chosen in view of the age, local, and general conditions.

3. '*Non-focal.*'—Without foci that can be seen in radiograms. In such cases in young children there is good hope of recovery with full movement after very long continued immobilization (*see Table I*).

*B.—Three Groups Distinguishable by the Age of the Patient:—*

1. Young children, 0–10, for whom prolonged immobilization is indicated, but neither diagnostic arthrotomy nor fusion.

2. The adolescents and adults, 15–50, for whom immobilization is preparatory to confirmation of diagnosis by arthrotomy, followed by fusion.

3. The elderly, from 50 onwards, for many of whom amputation is wiser than fusion.

**Diagnosis.**—*Provisional diagnosis*, coupled with immediate treatment, is the first step; after which for a time treatment and diagnosis run concurrently. In young children, indeed, a *clinical diagnosis*, carefully tested and reviewed, is sufficient.

Reasons are given for the conclusion that *diagnostic arthrotomy* is contra-indicated until the patient reaches an age when a positive diagnosis of tuberculosis will be quickly followed by fusion. The difficulties and risks of arthrotomy are discussed and a definition of its indications is suggested.

#### **Treatment.**—

**General.**—A combination of physical rest, metabolic stimulation by heliotherapy, well chosen and varied diet, and invigorating surroundings, serves to restore the vitality of the patient, and to raise it to a high level. This level should be maintained through the months or years of lymphatic disease and tuberculous bacillæmia. And the care must not be lessened or its period shortened because the knee has been fused.

**Local.**—There are only two safe end-results in tuberculosis of the knee—free movement and bony ankylosis.

The aim of local treatment, then, is the restoration of full movement; and, failing that, bony ankylosis. Immobilization is the paradoxical method of restoring movement.

Operative fusion is discussed, its rationale, its indications, its technical points, its contra-indications, and its failures. Figures are quoted. On the whole, it is highly successful; but should be avoided in those too young, too old, too ill, or obstinately septic.

A tuberculous sinus is a dangerous complication because its pyogenic infection is a disaster difficult to avoid.

Amputation has a restricted field as a life-saving measure for those disqualified for fusion by illness, age, or sepsis. Outside this field it is unanimously condemned.

Arthroplasty has no place in the treatment of tuberculosis of the knee or its results, for it is almost always either unsafe or impracticable.

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- <sup>1</sup> GHORMLEY, R. K., *Jour. Bone and Joint Surg.*, 1926, Oct., viii, 858.
- <sup>2</sup> WILLIS CAMPBELL, *Surg. Gynecol. and Obst.*, 1928, July, 89.

### THREE CASES OF TORSION OF AN APPENDIX EPIPLOICA OF THE SIGMOID COLON.

By G. H. COLT,

SURGEON, ROYAL INFIRMARY, ABERDEEN.

A CASE of this condition recorded recently by C. E. Taylor<sup>1</sup> and a recent case of my own reminded me of two other specimens which I had sent to the Royal College of Surgeons Museum. Sir Arthur Keith has very kindly forwarded to me the catalogue descriptions of the cases, and Mr. Sewell has drawn the specimens for reproduction, which are shown natural size.

In Dr. Taylor's case the patient, a married woman of 50, had suffered for three weeks from acute intermittent epigastric pain, increasing in severity and worse after meals. She often vomited after a meal. The bowels were usually regular, but diarrhoea had been noticed. There had been no melaena and no blood had been passed by the bowel. The abdomen was slightly distended, especially on the left side, and was tender in the right epigastrium, where an indefinite mass was palpable. Opaque-meal examination had demonstrated a slight spasm of the pylorus and dilatation of the coils of the small intestine. There had been a similar attack of pain five years previously. At the operation all the other abdominal organs were found normal, and torsion of a sigmoid appendix epiploica, which measured  $26 \times 20 \times 6$  mm., was found. It was twisted about a very narrow stalk and was a dusky purple colour. There was no inflammation of the surrounding peritoneum. It showed dilated veins on the surface, the vessels were congested, and one contained a clot. There was no inflammatory reaction. The little tumour was removed and the patient made an uneventful recovery.

The three specimens in the Museum of the Royal College of Surgeons are as follows:—

I. (6109.1, R.C.S. Museum Catalogue).—The vermiform appendix and an appendix epiploica which were removed by operation from the same patient. The appendix epiploica, which was attached to the sigmoid colon, is swollen and of a dull purple colour from hæmorrhage due to torsion of its pedicle. Near the apex of the vermiform appendix a small appendix epiploica has also undergone torsion. (*Fig. 378.*)

From a married woman, age 51, who had suffered for five years from increasing

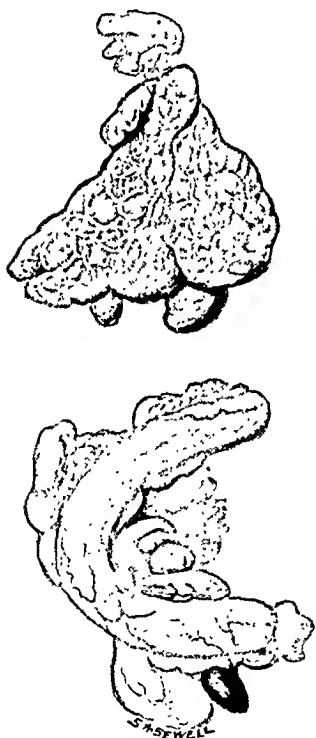


FIG. 378.

# TORSION OF APPENDIX EPIPLOICA

509

attacks of constipation, nausea, and occasional pain in the left iliac fossa. There had also been frequency of micturition dating from a confinement nine years previously. At the operation the abdominal and pelvic organs showed no pathological changes, except for those seen in the specimen. Radiological examination had shown no abnormality in the colon.

II. (6109.2, R.C.S. Museum Catalogue).—Two appendices epiploicæ adherent at their base, which were removed by operation from the sigmoid colon. One of them is partly of a deep purple colour due to hæmorrhage resulting from torsion. (Fig. 379.)

The patient, a woman, age 38, had undergone an operation four years previously for gangrenous appendicitis with abscess, which was drained. Since then there had been almost continuous pain in the left iliac fossa. During the last six months this had increased and was accompanied by attacks of constipation. There was no history of any acute initial attack. At the operation torsion of the appendix epiploica was found and two small bands of adhesions were present between the omentum and the lower part of the cæcum. The other abdominal organs were healthy.



FIG. 379.

III. (6109.3, R.C.S. Museum Catalogue).—An appendix epiploica with twisted pedicle, which was removed by operation from the middle of the anti-mesenteric border of the sigmoid colon. The pedicle consists of a narrow fibrous strand, not more than a millimetre in width at its distal attachment, and measuring  $\frac{3}{4}$  in. in length. The appendix itself is firm, of a dull ochre-yellow colour, with a few small hæmorrhages in its substance. Its surface is free from inflammation or adhesions. (Fig. 380.)

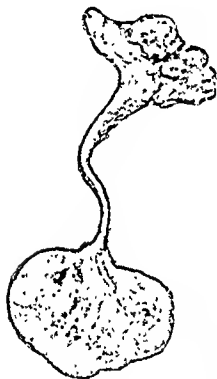


FIG. 380.

The patient, a man, age 64, had been well until the present illness. Four hours after lifting a heavy table in an awkward manner he was seized with sudden pain in the right side of the abdomen, and felt sick and vomited. The vomiting was repeated ten hours later. The pain was mainly in the loin and continuous, and tenderness was present there. A small quantity of blood was brought away from the bowel by an enema. The urine was normal. There was a slight rise of temperature ( $99^{\circ}$  F.). X-ray examination showed the absence of any calculus, but well-marked spondylitis of the lumbar vertebrae, and the fact that after operation there was a residual pain in the back and right loin for six days may have been evidence of the severity of the muscular strain. The diagnosis was subacute appendicitis. At the operation the vermiform appendix was found to be sufficiently inflamed to account for the symptoms. The twisted appendix epiploica was found as above mentioned and was removed. There was a marked terminal ileal kink. The descending colon was distended. The other abdominal organs were normal.

**Diagnosis.**—The condition would appear to be most common on the sigmoid, where appendices epiploicæ are most often seen. Though it might not be actually diagnosed, its presence might be strongly suspected if localized pain and tenderness with some functional disturbance of the bowel were present, and any gross lesion of the lumen, such as diverticulitis, were absent on radiological examination. In the four cases, the pain was intermittent and epigastric, with vomiting, worse after a meal, in one; in the left lower abdomen in two, with nausea and vomiting. The last patient, however, had strained his spine, which was already the site of osteo-arthritis.

More careful records in the future will no doubt help to elucidate the various points.

**Treatment.**—J. G. Knoflach<sup>2</sup> gives operation as the only treatment, and the degree of urgency appears from the severity of the case. He notes that care must be taken in respect of diverticulitis. In view of the discovery of loose bodies in the peritoneal cavity nature evidently sometimes takes the cure in hand.

### REMARKS.

The condition is certainly more common than would appear from the fact that Johanssen<sup>2</sup> in 1927 collected 20 cases from the literature. Others have told me of cases. Knoflach<sup>2</sup> mentions the formation of loose bodies by spontaneous amputation and the formation of cords which may cause ileus. The origin of the bean-like structures lying free in the pouch of Douglas and discovered accidentally at operation must have puzzled many surgeons. I can recall three instances. Their discovery should indicate a careful examination of the large intestine. The cause of the torsion is obscure. We know of no records of its following directly on deep abdominal massage or of its being common in those who do exercises for their abdominal muscles. Neither do we know that *la danse de ventre*, which might be expected to twist anything twistable, does more than give an occasional twist to the morals of some of the audience. Knoflach mentions inflammation of other abdominal organs and obesity. In the case from which Specimen II was taken the symptoms dated from an attack of gangrenous appendicitis. He says that a very fatty appendix or one affected by a tumour has been described several times. The double Specimen I must be excessively rare, and supports this connection. He gives inability to stand upright as an important symptom. The boundary to peritonitis may be crossed. It is clearly possible that the condition may be a cause of peritonitis, especially when diverticulitis has accompanied it, and should be looked for specifically in the absence of any other cause.

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<sup>2</sup> KNOFLACH, J. G., "Ueber intraabdominale Torsion des Netzes und der Appendices epiploiceæ", *Deut. Zeits. f. Chir.*, 1930, cxxv, 436.

SHORT NOTES OF  
RARE OR OBSCURE CASES

A LARGE MESENTERIC CYST IN AN INFANT  
SIX DAYS OLD.

By CECIL P. G. WAKELEY,  
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MESENTERIC cysts form a very definite group of cases, and have been recognized from the middle of the sixteenth century; up to the present

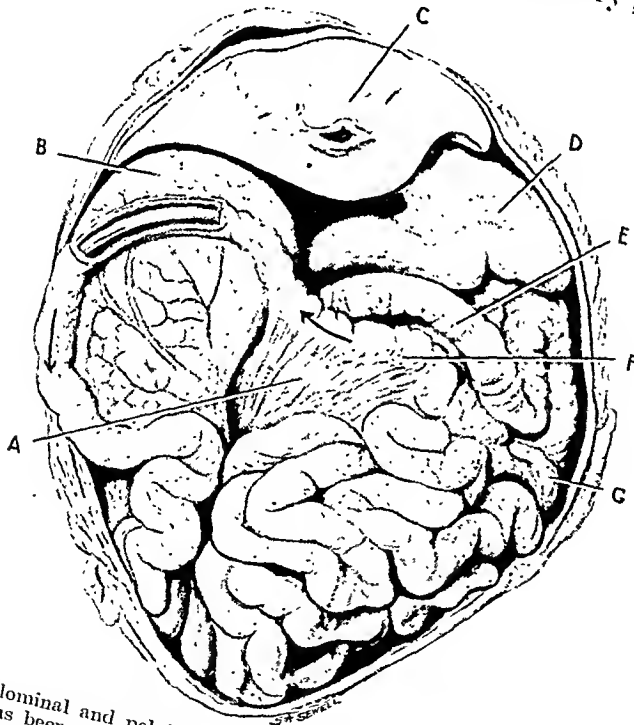


FIG. 381.—Abdominal and pelvic organs with part of the abdominal wall. The left half of the liver has been cut away. The mesenteric cyst can be seen lying in contact with the right lobe of the liver. A, Mesentery; B, Cyst; C, Liver; D, Stomach; E, Transverse colon; F, Jejunum; G, Appendix. (R.C.S. Museum: Specimen No. 6515.5.) ( $\times \frac{3}{8}$ .)

time some three hundred cases have been reported in the literature, the majority having been discovered at operation.



Although they have been described in the fœtus and in adults, the commonest age at which they give rise to symptoms is round about puberty. The terminal ileum is the usual site for these cysts, although they have been described in any part of the mesentery.

The following case is deemed of interest because it occurred in a male infant which died on the sixth day after birth, from bronchopneumonia.

**HISTORY.**—The infant was the second child; the labour was normal, and although the cyst was a large one, it did not seem to cause any obstruction during delivery. The child was fed on the breast from the second to the sixth day, and passed its motions quite regularly. It was not treated by any doctor, and was brought up to hospital because its mother stated it could not breathe. On admission to hospital it was found to be dead.

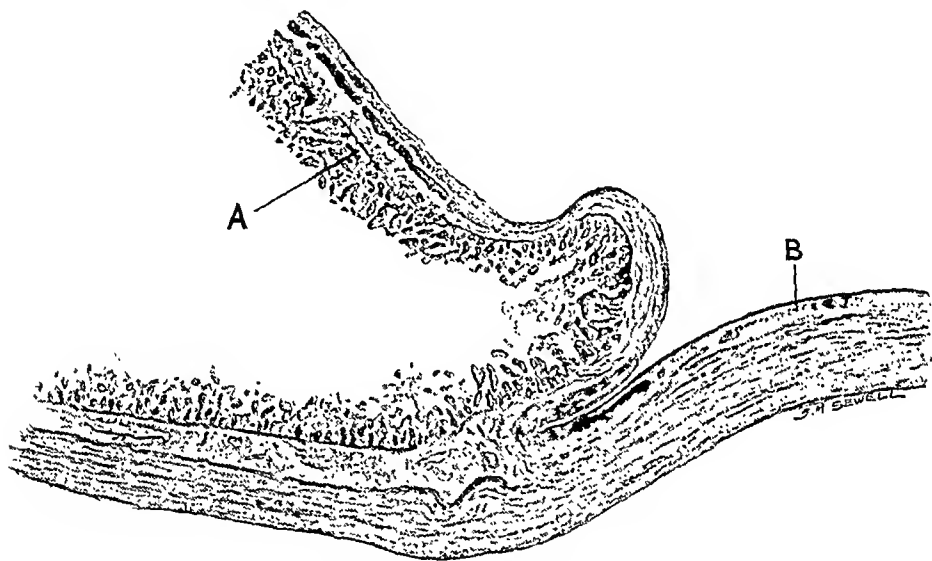


FIG. 382.—Microscopical drawing showing the junction of the cyst wall with the gut.  
A, Intestinal wall; B, Cyst wall. ( $\times 10$ .)

**POST-MORTEM.**—At autopsy, the cause of death was found to be bronchopneumonia; this was not to be wondered at, as the child was taken some six miles along the streets of London by its aunt on the day of its birth. The abdomen was considerably distended, and on opening it, the distension was found to be caused by a large mesenteric cyst lying on the right side in the mesentery of the proximal jejunum. The cyst measured  $2\frac{1}{2}$  in. in the chief antero-posterior diameter. It was connected with the concave border of the upper part of the jejunum and was enclosed within the layers of the mesentery (*Fig. 381*). The bowel was stretched over the upper surface of the cyst, but there was no communication between the two cavities. The cæcum and ascending colon were furnished with a distinct mesentery continuous with that of the ileum. Owing to the large size of the cyst, the cæcum and appendix were displaced towards the left iliac fossa. There was no block or atresia in any part of the alimentary canal.

Microscopical examination shows the cyst to be lined with a single layer of flattened oval or cubical cells, external to which is a layer of connective tissue, with scattered collections of lymphocytes. External to this layer is a broad layer of smooth muscle fibres, divided up in some places by strands of connective tissue; this layer is continuous with the circular muscle of the intestine, the two lying superimposed where the cyst and bowel are in contact (*Fig. 382*). External to the muscular layer is a narrow stratum of loose areolar tissue, continuous with the peritoneal covering of the bowel. The longitudinal muscle of the intestine is reflected on to the cyst wall for one or two millimetres; it then ceases abruptly, and is not elsewhere represented in the wall of the cyst.

The cyst contained a serous fluid in which was flocculent material consisting of granular matter and inflammatory and desquamated cells. It is apparently a lymphatic cyst of the submucosa of the intestine, which in enlarging has carried the circular muscle before it, while separating the longitudinal fibres.

The specimen is now in the Museum of the Royal College of Surgeons.

## GASTRO-PHOTOGRAPHY CONTROLLED BY POST-MORTEM EXAMINATION.

By J. R. M. WHIGHAM,

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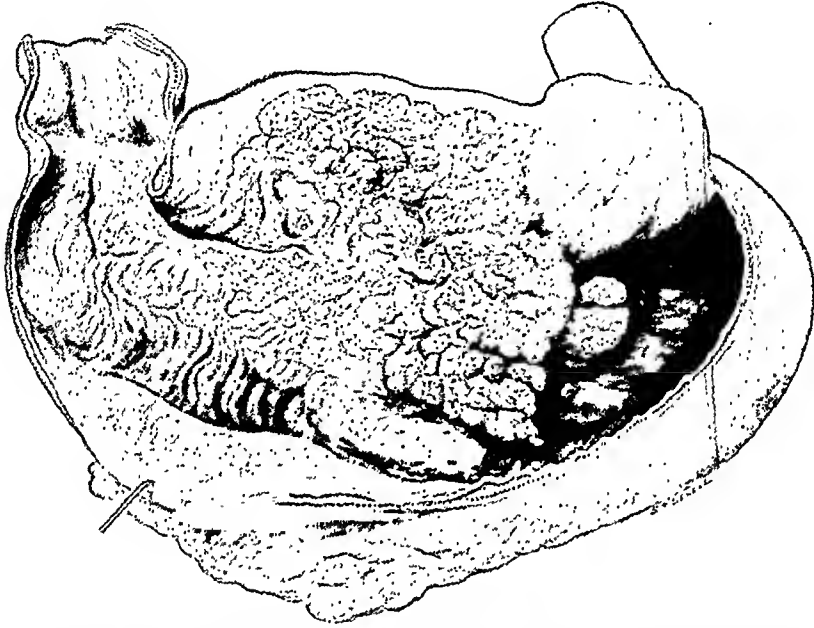
At a recent autopsy it was possible to compare a photograph of a carcinoma of the stomach taken during life with the findings present at death.

A man, R. H., age 67, was admitted to hospital on May 20. He had been ill with indigestion for about nine months, vomiting during the last month about half an hour after meals. There had been no hæmatemesis. On examination the patient was cachectic and a palpable mass was present in the epigastrium. The X-ray appearances following an opaque meal suggested the presence of carcinoma. On June 12 a gastro-photograph was taken, and the view of the anterior surface of the stomach showed the following appearances (*Fig. 383*). To the left there was a dark oval area surrounded by an irregular whitish margin, and to the right an area in which lighter shadows of varying intensity made up the field. The brightest of these were probably due to mucus.



FIG. 383.—Gastro-photograph of carcinoma of stomach. A, Edge of ulcer; B, Base of ulcer; C, Mucus; D, Papillomatous area.

Nine days afterwards, on June 21, the patient died, and at the post-mortem examination the stomach was found to be the seat of a large carcinoma (*Fig. 384*), presenting features very much resembling those seen in the gastro-photograph. The anterior wall of the stomach was covered with papillomatous projections which in one place, towards the pylorus, had under-



*FIG. 384.*—Post-mortem appearances. A, Area of ulceration ; B, Papillomatous growth ; C, Cut edge of anterior surface of stomach near greater curvature. The upper part of this surface has been turned upwards. ( $\times \frac{1}{2}$ .)

gone ulceration. In addition to the image of the growth, which is sufficiently striking, some accuracy of localization was also obtained, two considerations which should give encouragement for the future in this method of gastric investigation.

I am much indebted to Baron Von Veitschberger for assistance with the photography in this case.

REVIEWS AND NOTICES OF BOOKS.

*A Textbook of Surgery.* By JOHN HOMANS, M.D., Assistant Professor of Surgery. Compiled from Lectures and other writings of various members of the Surgical Department of the Harvard Medical School. Royal 8vo. Pp. 1195 + xii, with 513 illustrations. 1931. London: Ballière, Tindall & Cox. 40s. net.

THE most natural question to ask on meeting a new text-book of surgery is whether it can justify its publication as an addition to the large number already available. It may be said at once that this volume, compiled after eight years of strenuous effort by Dr. Homans, from the lectures and writings of a score of teachers in the Harvard Medical School, possesses certain features which amply justify its existence. Besides, it is a pleasant book to handle and to read, being printed in clear type on specially made paper.

At the present time a laudable and increasing interest is being shown in the history of science, and there are many who would welcome the introduction of lectures on the history of medicine into the course of study for a qualifying degree. A better plan still is to present each subject in its correct historical setting—and this is best done at the bedside or in the demonstration room—showing how the present position has been arrived at in each field. All will welcome the attempt made in this book to carry such a plan into practice, for each chapter begins with a short historical sketch which is not only of interest, but of great value to the student. The anatomy and physiology of each system is briefly but adequately described, and upon this foundation the pathological and clinical picture is built up. It will be seen that the work has been well conceived; and its real object—to record the teaching of surgery at the present day in the Harvard School—is in itself sufficient to recommend its careful study.

In order to limit the size of the book to one volume the author has decided to concentrate upon the practice of surgery, and to exclude the lengthy and often redundant chapters on pathology which the older books contain. In this we believe he has acted wisely, for now that the student has many works on pathology to refer to, he will be less than ever inclined to pay any attention to the pathological section in the surgery book, which is therefore better omitted. The present work contains just about as much pathology as is indispensable. The present work contains another feature which makes it particularly attractive is a bibliographical index, which, though it cannot be exhaustive, gives references to most of the important surgical literature, and is certain to supply not only information but also a stimulus to further study. It is essentially a students' text-book and not a book of reference, nor does its size permit of the inclusion of theoretical matter dealing with the many unsolved problems of surgery. It will therefore not be of real help to those studying for the higher surgical degrees and diplomas.

Some sections are particularly good, especially those on the nervous system and the chest; the greater part of the book is of the standard indicated above; and a few chapters are in comparison surprisingly poor. For instance, the descriptions of bone which are apparently of metabolic origin, all require revision. The accounts of most of the tumours, more especially of the salivary gland tumours, melanoma, hypernephroma, and tumours of the testis, are very sketchy and incomplete. The lack of discussion of subjects only partially understood is brought out when a giant-cell tumour of bone is described as an inflammatory condition, without giving the student any good evidence against its being a neoplasm. The genito-urinary system

as a whole gets less than its fair share of space, and diseases of the testis are practically omitted altogether. Such defects are so obvious that they will doubtless be attended to in future editions.

The book is illustrated by plentiful drawings, most of which give a clear picture of the subject in the text; but it is evidently difficult, if not impossible, to make a good drawing of a skigram, for most of the reproductions of X-ray pictures would be better omitted unless reproduced as photographs. It is natural to suppose that the book is intended primarily for Harvard men, and for medical students on the other side of the Atlantic. In spite of its many good qualities, it seems unlikely to replace the books to which students on this side have become accustomed.

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**On Faith and Science in Surgery.** By Sir JOHN BLAND-SUTTON, Bart., Vice-President and Consulting Surgeon to the Middlesex Hospital. Post 8vo. Pp. 109 + xii. Illustrated. 1930. London: William Heinemann (Medical Books) Ltd. 7s. 6d. net.

We all rejoice to see how gently time has dealt with Sir John Bland-Sutton; in this little volume he has published ten addresses delivered during the last five years in various cities of England, Scotland, Canada, and America. There is no sign of loss of vigour in his writing, nor has custom staled his infinite variety. Like all his writings, this book is charming: it should be read in a comfortable chair after a hard day's work; whether Sir John is talking of ligatures, of pearls and gall-stones, of cod-liver oil, of Jonah and the whale, or of the parasitic origin of man, the reader is held enthralled—"and still the wonder grew, that one small head could carry all he knew."

One of the joys peculiar to reading books by Sir John is the aptness of his quotations; we all know how fond he is of the Bible and Shakespeare; in the chapter on "Food and Physic" he quotes a very neat and appropriate couplet by Kipling:—

"Anything green that grew out of the mould  
Was an excellent herb to our fathers of old."

As in this and the preceding chapter, on "Faith in Drugs", he takes the opportunity of smiling at the prescriptions of the older physicians, we wonder he could resist the temptation of placing at the head of these—"I perceive that in all things ye are too superstitious."

The illustrations are excellent and add greatly to the pleasure of reading this book. We heartily commend it to our readers.

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**Modern Surgery.** By CHALMERS J. DACOSTA, M.D., LL.D., F.A.C.S., Samuel D. Gross Professor of Surgery, Jefferson Medical College; assisted by BENJAMIN LIPSHUTZ, M.D., F.A.C.S., Surgeon to the Mt. Sinai Hospital, Associate in Neuro-anatomy, Jefferson Medical College. Tenth edition, revised and reset. Royal 8vo. Pp. 1404, with 1050 illustrations, some in colours. London and Philadelphia: W. B. Saunders Co. 45s. net.

This may be described as a wonderful book, and the epithet may be applied to several of its features. In the first place, it is no mean achievement to have written a book of 1400 pages and to have carried it through several editions without assistance, even if we allow that in the preparation of the last two or three editions the author is much indebted to Dr. Lipshutz for the addition of new matter and very careful revision of former chapters; but it is even more wonderful when one realizes that it is nearly forty years since the book first appeared, and that, apart from having passed through so many editions, it has also been reprinted on innumerable occasions. The author's endeavour has apparently been to include everything possible, and not to exclude as much as he possibly can. Thus there is a long chapter devoted to the resuscitation of the asphyxiated and drowning, and a most fascinating account of the history of blood transfusion. Not only may it be regarded as a text-book of descriptive surgery, but all the operations in common use are described in detail, as are many which are more rarely performed. It is a remarkable compendium of surgical knowledge of which any surgeon, American or British, would be proud to be the author. Dr. DaCosta's energy and persisting enthusiasm

is all the more remarkable as he has just published a volume of selections from his papers and speeches, which range from "The Personal Side of Pepys" to "Medical Paris during the Reign of Louis Phillippe". His quotation on the fly-leaf of a charming verse from W. W. Story's *Contemporary Criticism* would at once disarm any reviewer who had critical intentions, but nevertheless, the present writer, claiming to be no less a lover of Dickens than Dr. DaCosta himself, must dissent from the misquotation of a paragraph most aptly introduced into the preface of this edition.

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**Operative Surgery: General and Special Considerations.** By Dr. MARTIN KIRSCHNER (Tübingen), authorized translation by I. S. RAVDIN, B.S., M.D. (Philadelphia). Super royal 8vo. Pp. 666 + xii, with 746 illustrations, mostly coloured. 1931. London: J. B. Lippincott Co. 50s. net.

THE drawings in this book are most artistic, and nearly all of them are coloured. It remains questionable, however, whether the illustrations are not overdone. Many of them are quite unnecessary, for methods and instruments are illustrated which the author confesses are of little value or out of date. Apart from this, it is a first-class book. Essentially one of technique, it is full of sound practical wisdom. The house surgeon will pick up from it many useful tips. Particularly well written is the chapter on the control of pain. All the more recent anæsthetics and the methods for their administration are described, and a very complete account of conduction anæsthesia is added. The chapters on hæmorrhage, shock, and transfusion are excellent. Clear indications are given of what constitutes a 'bad-risk' patient, and full instructions how these risks may be obviated by careful pre-operative investigation. The book is well translated.

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**The Management of Abdominal Operations.** By RODNEY H. MAINGOT, F.R.C.S., Surgeon, Royal Waterloo Hospital, London. Crown 8vo. Pp. 312 + xii. 1931. London: H. K. Lewis & Co. Ltd. 7s. 6d. net.

THE author of this book has undoubtedly filled a gap in surgical literature. It would have been better, in a work so full of views on which much criticism could be made, had he indicated the appropriate origin of such opinions under the proper chapter heading, instead of lumping his numerous co-editors together in the Preface. We should also have thought better of the book if the author had omitted a description of operations (many of them in our opinion inaccurate) and had concentrated on pre- and post-operative treatment.

Beginning with 'anæsthesia', he discusses in succeeding chapters (there are sixty-five) every possible kind of complication, and, in addition, gives an excellent account of pre- and post-operative treatment. Many pages are left blank for notes to be added by the reader.

The book is good in every way. It is a veritable 'gold mine' for a house surgeon or a senior student. It is a very useful reference book for an operating surgeon, and an excellent guide for the general practitioner and for nurses. The author has managed, by avoiding 'padding', to compress an enormous amount of information into singularly little space. He writes well and has chosen his co-editors with much judgement.

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**Abdominal Pain.** By JOHN MORLEY, Ch.M., F.R.C.S., Assistant Surgeon, Manchester Royal Infirmary, etc. With an Introduction by J. S. B. STOFFORD, M.D., F.R.S., Professor of Anatomy, University of Manchester. Medium 8vo. Pp. 191 + xvi, with 22 illustrations. 1931. Edinburgh: E. & S. Livingstone. 10s. 6d. net.

THE author's clinical investigations and papers upon the nature and significance of pain are well known. In this volume he reviews theories of the causation of abdominal pain which from time to time have held the field, and in particular closely examines the physiological basis of Mackenzie's theory, which was founded on the hypothesis of James Ross. After reviewing the contributions of Lennander, Hurst, Head, and others, the author then enunciates his own theory of abdominal pain. The

remainder of the book is taken up with observations upon the variations of pain in diseases and injuries of the different abdominal viscera and an endeavour to show that the author's hypothesis of the nature of abdominal pain is capable of closer correlation with clinical experience than any theory which has preceded it.

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*Chirurgie de l'Ulère gastrique et duodénal (Indications. Résultats).* By N. HORTOLMEI, Professeur de Clinique chirurgicale; and VL. BUTUREANU, Maître de Conférences, Faculté de Médecine de Jassy. Medium 8vo. Pp. 408, with 75 illustrations. 1931. Paris: Masson et Cie. Fr. 45.

THIS book will interest anyone engaged in gastric surgery. It contains an account of the various methods of surgical relief which have been devised and put into practice, with a critical review of the results as revealed by the literature of the subject, which has been very thoroughly searched. No one can read a book of this description without realizing that the final word on the surgery of the stomach has yet to be said. There are methods advocated by enthusiasts which have failed to gain recognition, such as Latarjet's denervation procedure, or are yet under trial, such as Babcock's cholecysto-gastrostomy recently revived by Braithwaite. Yet in the end the present-day problem resolves itself into that of deciding whether extensive gastroduodenal resections or some indirect method is preferable for gastric or duodenal ulcers. The natural dislike of the modern surgeon for resections for small pathological lesions is understandable and praiseworthy. But with our present ignorance of the etiology of gastric ulcer, it is at least a logical plan to ensure that conditions are so changed that gastric ulcer formation is almost impossible, even at the cost of the loss of a considerable part of the stomach. It is interesting, too, to observe how gastroduodenal resection is gradually making its way as a remedy for duodenal ulcer in France as well as in Central Europe. The authors of this book are frank advocates of large gastroduodenal resections, which they recommend should be carried out by the Reichei Pólya method, after which they declare they have never seen any serious trouble even after years have elapsed. They also strongly recommend some form of local as against general anæsthesia, finding spinal anæsthesia with novocain a satisfactory method. The drawback of its limited duration is removed by continuing with splanchnic and parietal infiltration when the operation lasts more than one to one and a half hours. They also follow the custom of Finsterer, unusual in this country, of draining the region of the parietal stump in every gastric resection performed by them. There is a very interesting chapter on the physiology of gastric resections, and the discussion of the many problems of gastric surgery is always well presented, with an appreciation of the essential problems.

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*Clinical Observations on the Surgical Pathology of Bone.* By DAVID M. GREIG, M.B., C.M., F.R.C.S.E., F.R.S.E., Conservator of the Museum of the Royal College of Surgeons of Edinburgh. Large 4to. Pp. 248 + xii, with 224 illustrations. 1931. Edinburgh: Oliver & Boyd. 30s. net.

THE author of this most interesting monograph is one of those who, from prolonged clinical experience combined with intensive study of pathology, have earned the right to generalize and formulate principles regarding bone disease. This treatise is attractive reading, being the work of an enthusiast as well as a keen observer, set out in admirable English. A motif runs through the book—it is that hyperæmia means bone absorption, excess of calcium and relative anæmia bone deposition or sclerosis. The work of Leriche and Policard is illustrated from all standpoints, and the researches of Dale and Lewis on histamine and acetylcholine in relation to vascular dilatation are pushed to the limits of their applicability. The illustrations are of a very high standard, and their value is greatly enhanced by the clinical histories which are supplied in the text.

Certain criticisms are called for. In some chapters the narrative proceeds from one subject to another without any headings to relieve the monotony of the text, the page headings being the only indication of the change. Very dogmatic views are expressed on controversial subjects without the furnishing of data in support

of them. One would have expected some photomicrographs or drawings to illustrate the conditions which are so minutely and confidently described. These criticisms are made because the book is so good that we could have wished it to be still more comprehensive. It should be read by all who interest themselves in surgery or pathology.

**Surgical Pathology of the Skin, Fascia, Muscles, Tendons, Blood, and Lymph Vessels.**  
By ARTHUR E. HERTZLER, M.D., Professor of Surgery, University of Kansas. Hertzler's Monographs on Surgical Pathology. Royal 8vo. Pp. 301 + xvi, with 260 illustrations. 1931. London: J. B. Lippincott Co. 21s. net.

THIS book is one of an ambitious series of nine monographs on surgical pathology, one of which, on the diseases of bone, has already appeared, the others being in process of preparation. The present volume is on conventional lines, the various affections being separately discussed, exemplified by an appropriate case, and accompanied by excellent illustrations and microphotographs. The space assigned to each condition has been carefully assessed, with the possible exceptions of Volkmann's ischæmia and Dupuytren's contracture, which, surely, in a work of this size, deserve more than the half page allotted to them. Omissions are few, the chief being an entire absence of any mention of idiopathic elephantiasis, that most curious of lymphatic diseases. The author's opinions on the etiology of the massive thickening of the deep fascia which is the invariable accompaniment of this condition would be of considerable interest.

Most of the theories advanced will be acceptable to the majority of British pathologists. The vexed question of the origin of the sebaceous cyst is discussed at considerable length, and Professor Hertzler, in pointing out that the cyst, when very small, is unattached to the skin, states his own belief that it arises from an abortive sacculation of the epidermis which has failed to develop into a sebaceous gland. With this, as with his somewhat arbitrary classification of the melanomas, one may be inclined to disagree, but the author's mature experience, and his masterly presentation of cases and researches, must inevitably command respect. The style is concise and pithy, making excellent reading, and altogether the book is worthy to rank with the best type of contemporary American writing.

**Medizinische Praxis Sammlung für ärztliche Fortbildung.** Edited by L. R. GROTE, A. FROMME, K. WARNEKROS. Vol. VII. Grundzüge der Neurochirurgie, by Prof. Dr. Med. WALTER LEHMANN (Frankfurt a. M.) Medium 8vo. Pp. 197 + xii, with 23 illustrations. 1930. Dresden and Leipzig: Theodor Steinkopff. Paper covers, 13.50; bound, RM. 15.

THIS book presents briefly, but most ably, an excellent summary of contemporary German thought on the pathology, diagnosis, and treatment of those nervous lesions which come within the scope of the surgeon. The author has taken into consideration also the views of the leading authorities in other countries. The sections are short, but are packed with information, and a very high standard is maintained throughout. The chapters on brain tumours, on spinal lesions, on the surgery of pain, and of the sympathetic nervous system are useful résumés of the work which neurological surgeons have to do. It is a book which can be dipped into with interest by everybody, and is particularly valuable to us in this country because it contains the views of German workers with full references to their original papers.

**La Sacro-coxalgie et son Traitement.** By PIERRE INGELSANS. Pp. 142, with 6 plates, Paris: Masson et Cie. Fr. 30.

A MONOGRAPH on tuberculous disease of the sacro-iliac joint, based on thirty-eight cases which are recorded in full. The author finds that diagnosis both by clinical methods and by radiographs is difficult. Most of the classical signs, such as that of Erichsen by compression of the pelvis, fail, and X-ray changes are often late. On the other hand, sciatica and localized pain and swelling over the joint posteriorly are usual. Abscess was present in twenty out of thirty-eight cases. The author advocates a fixation operation, using a graft from the posterior part of the ilium.



**Injuries and Sport. A General Guide to the Practitioner.** By C. B. HEALD, C.B.E., M.A., M.D. (Cantab.), M.R.C.P. (Lond.), Physician, with charge of Electrotherapeutic Department, Royal Free Hospital, etc. Demy 8vo. Pp. 543 + xxiv, with 380 illustrations. 1931. London: Humphrey Milford. Oxford University Press. 25s. net.

THE author has attempted a difficult task; he has endeavoured to give an account of the common injuries arising in sport of all sorts and to deal with their causation, pathology, and treatment, including under the last-named splinting, surgical intervention, and physical treatment. Dr. Heald's own work is concerned with the last named, and perhaps he would have been better advised to confine himself to his own specialty. His excursions into pathology and surgery are often open to criticism, his methods of splinting, as judged by the illustrations, are often crude, and the appliances illustrated inaccurate. For example, on p. 412 is an illustration of a "walking calliper for convalescent treatment of a fractured tibia". The appliance is not a caliper but a knee-cage with outside steel continued to the boot: it takes no bearing either on the head of the tibia or tuber ischii, and the side steel is on the wrong side if it is intended to act as a protection for non-union of the bone with a tendency to outward convexity. In fact, everything is wrong—including the spelling.

Dr. Heald quite rightly states that the diagnosis of an injury is only complete and efficient when it presents to the mind a true picture of the pathological condition of the injured part, but it is to be feared that this ideal is not carried out in the sections on special injuries. For example, he apparently still believes in the fable of the ruptured plantaris; Schlatter's disease is still to him a separation of the epiphysis of the tubercle of the tibia, and "progress must be watched by periodic X rays"—presumably to see the epiphysis reunite. Again, the importance of getting an accurate history of the exact mechanism of the production of an injury is not followed up in detail. The mechanism of production of dislocations of the semi-lunar bone and of fracture of the scaphoid in the wrist are not mentioned, and the occurrence of the latter as a handing-off injury at football is omitted. No mention is made of the frequent occurrence of strain of the internal lateral ligament as a skiing injury, and there are other examples of the omission of special games injuries of this sort, such as basal fracture of the terminal phalanx of the finger in wicket-keeping.

The book is well written and well illustrated. It gains interest by showing many well-known specialists in games in action photographs, and by illustrations of sports accidents. It will help the practitioner in the general diagnosis of injuries, but it is to be feared that the accounts of general mechanical and surgical treatment are not such as will commend it to the student or surgeon, and the methods of physical treatment described are really those of the specialist.

**Gonococcal Infection in the Male.** By A. L. WOOLBARST, M.D., Urologist and Director of Urological Clinics, Beth Israel Hospital, etc. Second edition, completely revised and enlarged. Medium 8vo. Pp. 297, with 140 illustrations, including 7 coloured plates. 1930. London: Henry Kimpton. 25s. net.

THIS monograph gives an excellent account of gonorrhœa and its complications in the male; it is clearly printed, very readable, and the illustrations are numerous, good, and help to elucidate the text.

We note with interest the author's personal conviction, set out in the preface to this edition, "that gonorrhœa must be considered a constitutional disease which can be cured only by the inherent reparative forces of the individual, acting through the blood-stream". This conviction is supported by a further statement of his that "the usual course of acute gonorrhœa is from eight to twelve weeks": and this in spite of his many years' experience as a urologist and of the almost innumerable methods of treatment that have been tried during the last thirty years. It really seems that the treatment of this disease has advanced little, if at all, since the days of Sir Henry Thompson. Indeed, the author seems to have abandoned the irrigation method of Janet and to have returned to the use of the syringe.

He considers that vasotomy (Belfield's operation) is the most valuable modern

advance in the management of gonococcal arthritis, and that this is the one possible way of reaching the vesiculæ seminales with salts of silver or other injections: judging, however, from his illustration of the anatomy of these structures, it would seem probable that the fluid might run straight into the prostatic urethra without filling the vesiculæ.

The chapter on sexual neuroses following gonorrhœa is interesting; the author appears to consider that they are due to local lesions in the prostatic urethra combined with a 'neurasthenic soil'; he advocates local treatment by diathermy, etc., through a urethroscope.

The author is to be congratulated on having brought this second edition of his book so fully up to date.

## BOOK NOTICES.

*[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.]*

**Modern Proctology.** By MARION C. PRUITT, M.D., F.R.C.S.E., F.A.C.S., Associate in Surgery, Emory University School of Medicine, etc. Super royal 8vo. Pp. 404, with 233 illustrations. 1931. London: Henry Kimpton. 36s. net.

**Minor Surgery.** By LIONEL R. FIFIELD, Late Surgical First Assistant and Registrar, London Hospital. Second edition, revised by R. J. McNEILL LOVE, M.S. (Lond.), F.R.C.S., Surgeon, the Royal Northern Hospital, etc. Crown 8vo. Pp. 440 + viii, with 281 illustrations. 1931. London. H. K. Lewis & Co. Ltd. 12s. 6d. net.

**Emergency Surgery.** By HAMILTON BAILEY, F.R.C.S., Surgeon, Royal Northern Hospital. Vol. II. Thorax, Spine, Head, Neck, Extremities, etc. Large 8vo. Pp. 415 + xviii, with 430 illustrations, some in colour. 1931. Bristol: John Wright & Sons Ltd. 25s. net.

**Die Schlemhaut des Verdauungskanaals im Röntgenbild.** By HENRI CHAUL and ALBERT ADAM, with a Foreword by FERDINAND SAUERBRUCH. Imperial 8vo. Pp. 229 + viii, with 219 illustrations. 1931. Berlin and Vienna: Urban & Schwarzenberg. Paper covers, RM. 22.50; bound, RM. 25.

**Chirurgische und konservative Kosmetik des Gesichtes.** Edited by Dr. LEANDER POHL (Vienna). Large 8vo. Pp. 383 + viii, with 445 illustrations and 3 coloured plates. 1931. Berlin and Vienna: Urban & Schwarzenberg. Paper covers, RM. 25; bound, RM. 28.

**Surgical Pathology of the Genito-urinary Organs.** By ARTHUR E. HERTZLER, M.D., Professor of Surgery, University of Kansas. Hertzler's Monographs on Surgical Pathology. Royal 8vo. Pp. 286 + xviii, with 222 illustrations. 1931. London: J. B. Lippincott Co. 21s. net.

**Atlas de Radiographie osseuse. I. Squelette normal.** By G. HARET, A. DARIAUX, Electro-radiologistes des Hôpitaux de Paris; and JEAN QUÉNU, Professeur agrégé à la Faculté de Médecine, Chirurgien des Hôpitaux de Paris. With the collaboration of H. P. CHATELLIER, Oto-rhino-laryngologiste des Hôpitaux. Second edition, revised and enlarged. Preface by PIERRE DUVAL. Large 4to. Pp. 186, with 149 illustrations. 1932. Paris: Masson et Cie. Fr. 200.

**Atlas de Radiographie osseuse. II. Pathologie.** In two volumes. Vol. I. Lésions traumatiques. By G. HARET and A. DARIAUX, Electro-radiologistes des Hôpitaux de Paris; and JEAN QUÉNU, Professeur agrégé à la Faculté de Médecine, Chirurgien des Hôpitaux de Paris. Vol. II. Lésions non traumatiques. By ETIENNE SORREL, Chirurgien de l'Hôpital Trousseau; and Mme. Y. SORREL-DEJERINE, Ancien Interne des Hôpitaux. Large 4to. Pp. 344, with 897 illustrations. 1931. Paris: Masson et Cie. In two volumes, Fr. 310; in one volume, Fr. 280.

**Bendien's Diagnostic Methods for Cancer and Principles of Treatment.** By A. A. MILLER, M.D. With an Introduction by Dr. S. G. T. BENDIEN. Crown 8vo. Pp. 80, with 9 illustrations. 1931. London: H. K. Lewis & Co. Ltd. 3s. 6d. net.

**Guy's Hospital Reports.** Edited by ARTHUR F. HURST, M.D. Vol. LXXXI (Vol. XI, Fourth Series). No. 3. July, 1931. Royal 8vo. Pp. 253-378. Illustrated. 1931. London: The Lancet Ltd. Single numbers, 12s. 6d. net; annual subscription, £2 2s. net.

- Tumours of the Breast: their Pathology, Symptoms, Diagnosis and Treatment.** By Sir G. LENTHAL CHEATLE, K.C.B., C.V.O., F.R.C.S., Consulting Surgeon and Emeritus Lecturer on Surgery, King's College Hospital, London; and MAX CUTLER, B.Sc., M.D., Director of Tumor Clinic, Michael Reese Hospital, Chicago. Large 8vo. Pp. 596 + viii, with 468 illustrations and 18 coloured plates. 1931. London: Edward Arnold & Co. 50s. net.
- Division palatine: Anatomie—Chirurgie phonétique.** By VICTOR VEAU, Chirurgien de l'Hôpital des Enfants assistés, with the collaboration of Mme. S. BONEL. Large 8vo. Pp. 568 + viii, with 786 illustrations. 1931. Paris: Masson et Cie. Fr. 140.
- Traitement du Bec-de-lièvre unilatéral (Procédé du Dr. Veau).** By PAUL PLESSIER, Ancien Interne des Hôpitaux de Paris. Large 8vo. Pp. 148 + iv, with 106 illustrations and 16 plates. 1931. Paris: Masson et Cie. Fr. 30.
- Thomson and Miles' Manual of Surgery.** By ALEXANDER MILES, M.D., LL.D., F.R.C.S.E., Consulting Surgeon, Royal Infirmary, Edinburgh; and D. P. D. WILKIE, M.D., F.R.C.S.E., Professor of Surgery, University of Edinburgh. Eighth edition. Crown 8vo. Vol. II. Extremities, Head and Neck. Pp. 685 + viii, with 303 illustrations. Vol. III. Thorax and Abdomen. Pp. 578 + xii, with 177 illustrations. 1931. London: Humphrey Milford. 12s. 6d. net each.
- The Laboratory in Surgical Practice.** By C. E. DONNS, M.V.O., M.D., Courtauld Professor of Biochemistry in the University of London, etc.; and LIONEL E. H. WHITBY, C.V.O., M.D. (Camb.), M.R.C.P. (Lond.), D.P.H., Bacteriologist to the Middlesex Hospital, Bland-Sutton Institute of Pathology, etc. Demy 8vo. Pp. 187 + x. Illustrated. 1931. London: Constable & Co. 8s. 6d. net.
- La Pratique chirurgicale illustrée.** By VICTOR PAUCHET. Fasc. XVII. Super royal 8vo. Pp. 268 + vi, with 186 illustrations by S. Dupret. 1932. Paris: Doin et Cie. Fr. 65.
- Lehrbuch der Röntgendiagnostik.** By H. R. SCHINZ, W. BAERNSCH, and E. FRIEDL, with the collaboration of M. HOLZMANN, A. HOTZ, O. JÜNGLING, E. LIEBMAN, E. LOOSER, and K. ULRICH. Third edition. Two volumes. Imperial 8vo. Pp. 1623 + xxviii, with 2714 illustrations and 5 photographic plates. 1932. Leipzig: Georg Thieme. Paper covers, M. 214; bound, M. 220.
- Indikation und Technik des künstlichen Pneumothorax.** By Dr. HEINRICH MAYRHOFER (Vienna). Large 8vo. Pp. 35, with 33 illustrations. 1932. Vienna: Julius Springer.
- Chirurgische Indikationen für Ärzte und Studierende.** By Prof. Dr. KARL RESCHKE (Griefswald). Vol. I. Super royal 8vo. Pp. 357 + viii. 1932. Berlin: F. C. W. Vogel. Paper covers, RM. 22; bound, RM. 24.
- Fractures of the Jaws.** By ROBERT H. IVY, M.D., D.D.S., F.A.C.S., University of Pennsylvania; and LAWRENCE CURTIS, A.B., M.D., D.D.S., University of Pennsylvania. Medium 8vo. Pp. 180 + viii, with 177 illustrations. 1932. London: Henry Kimpton. 21s. net.
- Matas Birthday Volume. A Collection of Surgical Essays written in Honor of Rudolph Matas, New Orleans.** Super royal 8vo. Pp. 396 + xiv. 1931. New York: Paul B. Hoeber, M.C. \$10.00.
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## *SOME BYGONE OPERATIONS IN SURGERY.*

By SIR D'ARCY POWER, K.B.E., LONDON.

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### VIII. THE FIRST LOCALIZED CEREBRAL TUMOUR.

DELIBERATE brain surgery for exploration purposes is of such recent origin that the great Index Catalogue of the Surgeon-General's Library at Washington contains no mention of it in the volume issued in 1881. The subject, however, was attracting attention. David Ferrier published the *Functions of the Brain* in 1876, and in 1878 delivered the Goulstonian Lectures at the Royal College of Physicians of London, taking as his subject, "The Localization of Cerebral Disease". His facts were obtained by experiments conducted for the most part on dogs and monkeys, but no surgeon utilized them until Nov. 25, 1884. On Nov. 3 in that year a patient was admitted into the Hospital for Epilepsy and Paralysis in Regent's Park under the care of Dr. Alexander Hughes Bennett (1848-1901), who was then Assistant Physician to Westminster Hospital. Dr. Bennett was the son of a well-known Edinburgh physician, and himself died of some obscure disease of the nervous system after twenty years of almost continuous pain.

The patient was a farmer aged 25 whose chief complaint was paralysis of the left hand and arm which prevented his working. He said that about four years previously, while in Canada, a piece of timber fell from a house, struck him on the left side of the head, and knocked him down. He lost consciousness for a few moments, but soon recovered and went on with his work. He was quite well on the following day, and, with the exception of occasional headaches, remained in good health for a year. He then began to feel a twitching in the left side of his mouth and tongue. This soon developed into paroxysmal attacks, and some months afterwards he had a 'fit', which began with a peculiar feeling in the left side of the face and tongue, and turning of the head also to the left. The sensation ran down the left side of the neck to the arm and leg, ending in loss of consciousness and general convulsions. The attacks increased in frequency and severity until, in August, 1884, the patient was no longer able to go on with his work.

Examination showed him to be in robust general health with unimpaired intelligence. He suffered from frequent and violent attacks of stabbing pain

in the head, which was diffused over the vertex. Deep pressure elicited tenderness when it was made over the parietal region near the right of the sagittal suture on a level with a line drawn vertically from the anterior portion of the external auditory meatus. The movements of the eyeballs and pupils were normal. The patient could read No. 3 of Jaeger's types at twelve inches with his left eye, and No. 5 with his right. Examination of the fundi showed the usual appearances of optic neuritis, more marked on the right, the retina of which had numerous minute hæmorrhages. The left side of the face was slightly immobile, and the tongue pointed a little to the left when it was protruded. There was complete paralysis of the left fingers, thumb, and hand. The movements of the elbow-joint were very limited and those of the shoulder impaired. There was no attempt at pronation or supination of the forearm. The muscles were neither rigid nor wasted. There was slight lameness on walking because the toes did not completely clear the ground. The knee-jerk was greater on the left than on the right side.

Observation in hospital showed that the patient often suffered from paroxysmal attacks of lancinating pain in his head, sometimes lasting for twelve hours. These attacks of pain were so violent that the patient was occasionally delirious and kept the whole ward disturbed by his cries. He also suffered from severe vomiting, which often lasted several days. The paroxysmal twitchings took place many times a day, and consisted of arrhythmical tremor which began in the first, second, and third fingers of the left hand, spreading afterwards to the thumb and wrist as high as the elbow. This continued perhaps for a minute and then ceased, generally by the thumb being held or rubbed. Another form began in the left angle of the mouth and side of the face, with a feeling as if the tongue was being contracted. Sometimes, but not usually, the movements began in the face and arm, extended from one to the other, and from thence down the side of the neck and body to the leg, so that the whole left side was convulsed without any loss of consciousness. The diagnosis given was "an encephalic growth probably of limited size involving the cortex of the brain and situated at the middle part of the fissure of Rolando".

The risks of an operation were fully explained to the patient and his friends, who readily consented to its being undertaken. The patient was anesthetized on Nov. 25, 1884, in the presence of Dr. Hughes Bennett, Dr. Hughlings Jackson, Dr. David Ferrier, and others. The operation was performed by Mr. Rickman Godlee (1849-1925), who had been Assistant Surgeon at University College since 1877 and was also Senior Demonstrator of Anatomy in the medical school under Professor G. Viner Ellis.

The head of the patient had been shaved in the ward except the very lowest part of the scalp quite below the occiput, the whole was soaked with carbolic acid lotion (1-20), but no particular attention was paid to some sore places left after the application of blisters to the upper part of the neck—indeed, these sore places were not noticed until after the operation. The parts were again washed with 1-20 carbolic lotion after the patient had been placed upon the operating-table, and the upper part of the body was then surrounded by carbolized towels, one being placed beneath the head. Instruments and hands were soaked in the same lotion, and the spray was used

throughout the operation. A 1-in. trephine was applied  $1\frac{1}{4}$  in. from the middle line and  $\frac{1}{2}$  in. behind a line drawn vertically from the meatus of the right ear over the point of maximum tenderness. The dura mater was found normal in appearance. A crucial incision was made in it, and the brain substance bulged through the cut, as was thought, abnormally. The surface



RICKMAN J. GODLEE, AGED 25.

appeared somewhat more yellow in colour than natural, but seemed otherwise to be healthy. A second trephine opening was then made slightly in front of and overlapping the first, and the angles thus left were rounded off with a chisel and hammer, the brain being protected by a copper spatula. The incision in the dura mater was prolonged, exposing an increased surface of

brain, but without further revelations. The trephine was applied a third time so as to join the two former openings posteriorly, and when the edges were chipped off a triangular aperture with rounded angles was left, measuring 2 by  $1\frac{3}{4}$  in. The incision in the dura was then prolonged, exposing a surface of brain nearly the size of the opening in the skull, which presented the same appearance as that already described. Occupying most of this space, and crossing it obliquely from above and behind, forwards and downwards, was a large convolution, along the posterior aspect of which ran a large blood-vessel. An incision was made into the centre of this convolution, and from  $\frac{1}{8}$  to  $\frac{1}{4}$  in. below the surface a large transparent lobulated solid tumour was seen, thinly encapsulated but perfectly isolated from the surrounding brain substance. The surface and sides of the growth were easily separated by means of a narrow spatula of steel so tempered that it could be bent into any shape required. After the superficial portion of the tumour was isolated the finger, was, as far as possible, inserted behind the tumour and attempts were made to enucleate it, but in doing so the upper half broke across. A large Volkmann's sharp spoon was then employed to scrape out the deeper parts of the growth, and this was continued until all the morbid material was removed. No artery of any size spouted, but there was a rapid and troublesome oozing of blood, which accumulated quickly as soon as the sponge was removed.

The cavity left was about  $1\frac{1}{2}$  in. in depth and of a size into which a pigeon's egg would fit. The hæmorrhage was arrested by applying over the cut surface a suitable electrode from an electro-cautery. The dura mater was then drawn together by a few carbolized silk sutures, and a drainage tube of indiarubber was inserted into the wound beneath the dura mater. The skin was brought together accurately by sutures of silver wire and silk. "The carbolic spray was used during the entire operation and, both before and after, all the ordinary antiseptic precautions were taken". The operation lasted two hours. Subsequent examination proved the tumour to be a glioma about the size of a walnut.

The patient did well for the first three days, but when the dressings were changed on the fourth day the wound was found to be swollen and the discharge had a decidedly putrefactive smell. A cerebral hernia as large as half an orange had developed on the fifth day, and the patient died on Dec. 23—twenty-eight days after the operation, as the report states, "not from any special failure of the nervous system, but from the effects of a secondary surgical complication".

The operation naturally excited great interest, not alone in professional circles. *The Times* published two sensible leading articles on it which appeared on Dec. 16 and 27, 1884, the latter after a somewhat acrimonious and ill-informed correspondence on the part of the Antivivisectionists.

The portrait of Sir Rickman Godlee is made from a photograph kindly lent by Lady Godlee, who believes that it was taken about 1875 when he was aged 25.

**DIAPHRAGMATIC HERNIA.**

BY J. BASIL HUME,

ASSISTANT SURGEON. ST. BARTHOLOMEW'S HOSPITAL, LONDON.

*(Being a Hunterian Lecture delivered at the Royal College of Surgeons of England on January 21, 1931.)*

DEFECTS in such an important structure as the diaphragm cannot fail to have attracted the attention of our honoured patron whose name is commemorated by the Hunterian Lectures. In those writings which have come down to us there is no mention of any observations on diaphragmatic hernia, but the scattered notes collected by Richard Owen, and the unwritten evidence on the galleries of the Hunterian Museum, show clearly how he observed the condition of the diaphragm in the higher reptiles and in birds, and how he must have compared this with the completed membrane found in mammals. Hunter must have seen cases of traumatic diaphragmatic hernia during his service with the army in Belle Isle and in Portugal in 1761 and 1762, as penetrating wounds of the diaphragm were commonly inflicted by the sword and bayonet. He was also probably familiar with the work of Petit, and with the monograph of Morgagni on the subject published in 1769.

The condition is sufficiently unusual to excite the attention of physician, surgeon, anatomist, and radiologist alike, and in consequence a very large proportion of the cases encountered have been recorded. My reason for continuing along so well-trodden a path is a desire to present an anatomical and pathological picture of the different varieties of the condition and their mode of production.

**Classification.**—A very simple classification will suffice: (1) Congenital. (2) Acquired: (a) non-traumatic, (b) traumatic.

**CONGENITAL DIAPHRAGMATIC HERNIA.**

This may be defined as a protrusion of some of the abdominal contents through a congenital defect in the diaphragm. In the majority of cases such a defect must be produced by a failure of union of the various parts of the diaphragmatic primordium, and the herniated structures will lie freely in the pleural cavity uncovered by any hernial sac. In a smaller number of instances the membranous diaphragm may have been completed, but some temporary rise of intra-abdominal pressure in the embryo may bring about a hernia covered by a hernial sac. This is only likely to occur before the muscular invasion of the diaphragm is complete. For a clearer explanation of this and of the possible defects and malformations of the diaphragm we must turn to its embryology.

**Embryology.**—In the earliest stages of development the coelomic cavity is not divided in any way. At its anterior and cephalic extremity the heart

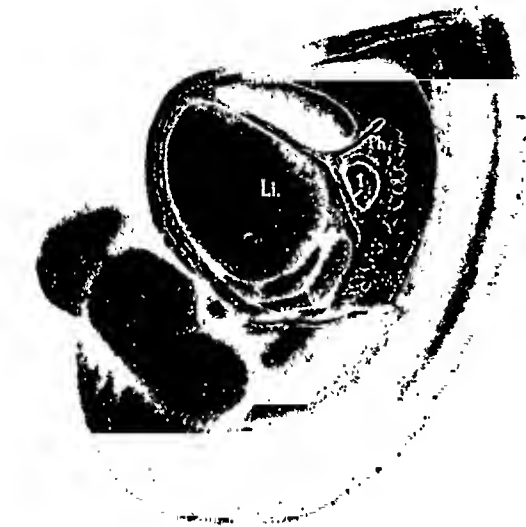


is gradually taking shape, supported on its inferior and dorsal surface by a mass of mesoderm known as the 'septum transversum'. This at first

stretches from side to side towards the cranial part of the coelom, but later recedes to a more caudal level and lies obliquely. Running into the septum transversum on either side are the ducts of Cuvier, and as the septum takes up its oblique position these form well-marked ridges on the lateral walls of the coelom, known as the 'pleuro-pericardial ridges' (*Fig. 385*). They ultimately become incorporated with the dorsal mesocardium, assisting in the formation of its dorsal wall.

Running down through the septum transversum and the pleuro-pericardial ridges are the phrenic nerves, which at their point of entrance into the diaphragm mark the dorsal limit of the portion developed from the septum.

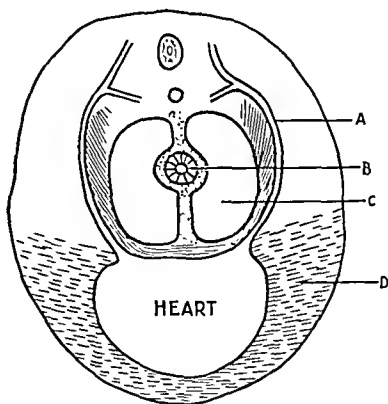
dorsal mesentery of the foregut



**FIG. 385.**—Reconstruction of 11-mm. embryo, showing the position of the septum transversum and the pleuro-pericardial and pleuro-peritoneal ridges. The arrow passes through the pleuro-peritoneal hiatus. Li., Liver; 1., Lung; Ph., Phrenic nerve; S., Stomach; W., Wolffian body. (*After Keibel and Mall.*)

In the mid-line dorsally and caudally the connects the dorsal wall of the embryo, in the aortic region, with the septum transversum where this has the foregut resting on it (*Fig. 386*). The liver bud grows from this foregut directly into the septum, and subsequently the foregut draws away from the septum, where the latter lies on its caudal aspect.

Dorso-laterally two ridges on the lateral wall mark out the space on this wall which corresponds with the upper surface of the septum transversum as seen in the floor of the pleuro-peritoneal passage. The more cranially situated one is the pleuro-pericardial ridge already mentioned; the caudal one shows as a sort of hepatic suspensory band to the septum and becomes more marked as the liver grows. This latter fold marks the edge of the structure which becomes the pleuro-peritoneal membrane as the pleural cavity extends caudally.



**FIG. 386.**—Diagrammatic cross-section showing the position of the pleuro-peritoneal passage. A, Duct of Cuvier; B, Foregut; C, Pleuro-peritoneal passage; D, Septum.

Thus an open passage to the abdomen is bounded dorso-laterally by this caudal suspensory fold, and ventro-medially by the septum and its continuity with the mesentery round the foregut. The hiatus is gradually narrowed as the pleuro-peritoneal folds extend, assisted by the dorso-lateral extension of the liver, and these folds can be seen in an 11-mm. embryo running into the septum transversum at the point of junction with the dorsal mesocardium. An opening, however, remains for some time dorso-laterally, and here the cephalic end of the suprarenal lies half in the pleural and half in the peritoneal cavity. The opening is ultimately closed in the seventh or eighth week of intra-uterine life by arching of muscle cells over the upper end of the suprarenal and the descent of that body. The left hiatus is said to close later than the right. Brachet has argued that as the pleuro-peritoneal fold is anchored to the mesonephros the descent of the latter draws the fold down with it.

Ventrally the membranous diaphragm is completed by an extension forwards from the septum transversum, due to a growth of the septum accompanying the advance of the liver bud in the ventral mesentery towards the opening of the umbilical stalk. A separation occurs subsequently between the anterior surface of the liver and the diaphragm.

As early as the fifth week the elements of the membranous diaphragm are invaded by muscle cells presumably originating in the cervical region. These spread out from the centre of the diaphragm to the periphery, and the central tendon is formed as a result of the disappearance or degeneration of these muscle cells. The last portion to be invaded is the lumbo-costal triangle, marking the final point of closure of the hiatus. (Fig. 387.)

**Types of Congenital Diaphragmatic Hernia.**—Herniæ which are undoubtedly congenital in origin are found in the region of the lumbo-costal triangle, in the dome of the diaphragm, and in the œsophageal orifice. They may be classified as follows: (1) Hernia through the pleuro-peritoneal hiatus. (2) Hernia through the dome. (3) Hernia through the œsophageal orifice: (a) thoracic stomach, (b) para-œsophageal hernia.

#### Causation of Congenital Diaphragmatic Hernia.—

1. **HERNIA THROUGH THE PLEURO-PERITONEAL HIATUS.**—This is due to the failure of closure of the median and dorsal portion of the pleuro-peritoneal membrane. The passage remains open until the third month of intra-uterine life, that on the left side possibly closing later than the right.

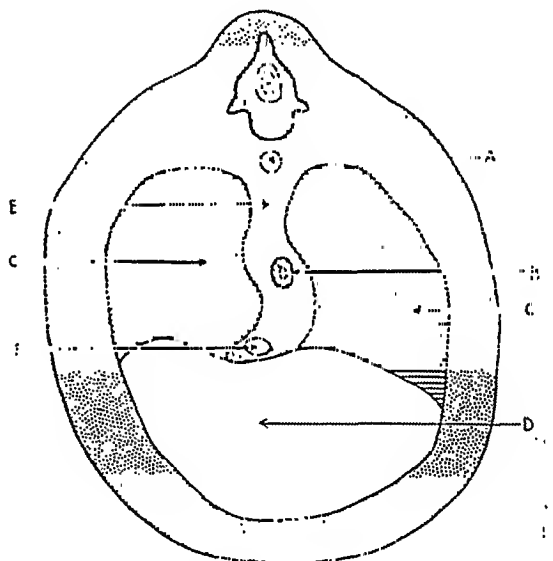


FIG. 387.—Diagram showing contributions to the Aorta; B, Œsophagus; C, Dorsal mesentery; D, Septum transversum; E, Dorsal mesentery; F, Inferior vena cava.

The hiatus is situated postero-laterally between the lumbar and costal fibres of the diaphragm, its position being indicated in the normal diaphragm by a crossing of the muscular fibres in front of the arcuate ligament, an area

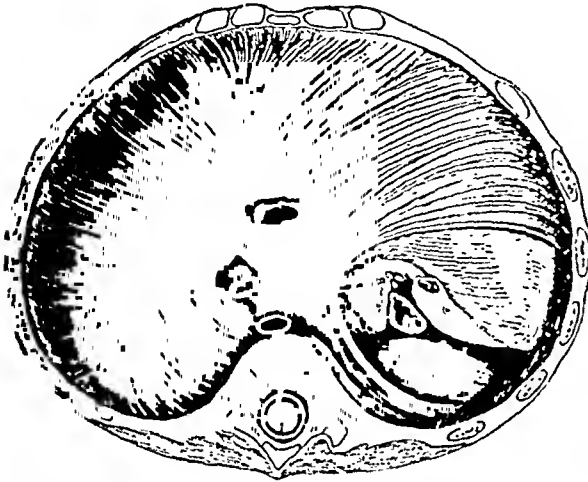


FIG. 388.—A diaphragm showing from the thoracic aspect the position of a hiatal defect.

known as the 'lumbo-costal triangle'. Considerable variation is found in the size of hiatal defects (*Figs. 388, 389*). Some may pass outwards to the thoracic parietes, and some forwards into the region of the septum transversum. An exaggerated form is seen when the left half of the diaphragm is absent.

In the majority of cases when a hiatal defect is present the greater part of the intestine lies in the pleural cavity. The large intestine lies to the left of the small intestine in the position occupied before

rotation of the gut has taken place. The stomach is sharply bent over to the left and drawn up into the thorax, and there is no hernial sac. The condition in its extreme form is incompatible with extra-uterine life of more than a few weeks' duration.

Consideration of the position of the developing intestine shows that herniation must take place at the time of the return of the intestine from the umbilical stalk, which it has occupied until the end of the second month (*Fig. 390*). Prior to this time there is no mobile portion of the intestine in the abdominal cavity, but a sudden return takes place owing to the fact that the liver edge recedes from the orifice of the umbilical stalk and there is a decrease of intra-abdominal pressure. This is the most likely time for herniation to occur.

It must be remembered that the pleuro-peritoneal passage is small at the time when the hernia occurs, and bears in a 20-mm. embryo about the same relation to the size of the rest of the abdominal wall as the orifice of a large inguinal hernia does in an adult. The presence of a hernia in the opening will not only prevent closure taking place, but the herniated structures may

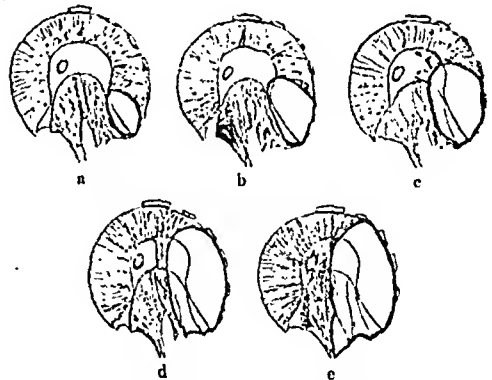


FIG. 389.—A series of diaphragms showing the variations in the extent of the hiatal defects.

so press upon the diaphragmatic primordium as to cause either displacement or pressure atrophy.

A very important contributory factor is delay in the development of the liver. It is almost certain that the growth of the right lobe of the liver brings about an earlier closure of the right hiatus. Up to the fifth or sixth week the lobes of the liver are fairly symmetrical, but after that period the right lobe grows more rapidly than the left, which extends dorso-laterally into the pleuro-peritoneal fold.

## 2. HERNIA THROUGH THE DOME.—

The explanation of the production of a hernia in this situation is not nearly so obvious as in the type just described, and presents a much more complex problem. Had this condition not been discovered in foetuses at the time of birth it might be doubted whether a congenital hernia could occur in the dome of the diaphragm at all, but fortunately sufficient cases have been recorded to dispel this doubt. My own view is that such a congenital hernia does occur, usually without a hernial sac, and that it is due to rupture or destruction

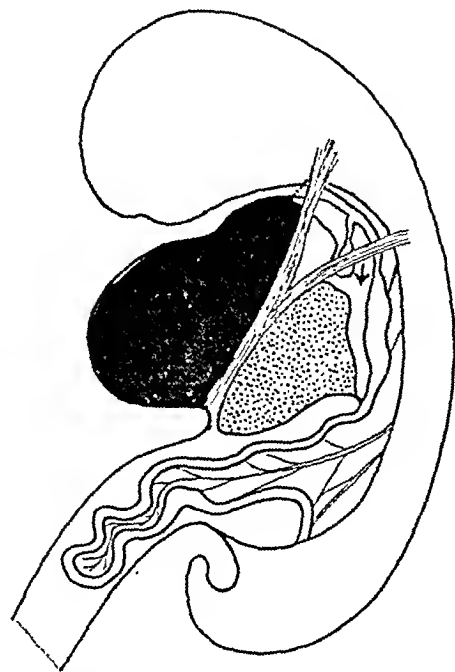


FIG. 390.—Diagram of an 11-mm. embryo, showing the extent of the coelomic cavity (blue), the retro-pericardial situation of the stomach, and the intestine in the umbilical stalk.

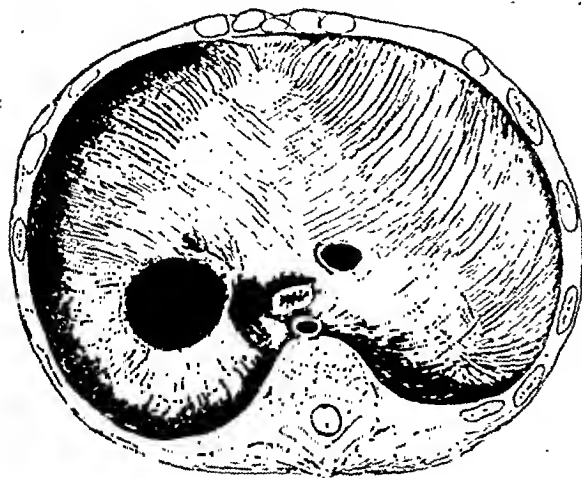


FIG. 391.—A diaphragm showing from the thoracic aspect a defect in the left dome.

of a portion of the membranous diaphragm.

A defect is more common in the left dome than in the right, which is readily accounted for by the protective effect of the larger right lobe of the liver. The position of the defect is fairly constant, though there is considerable variation in its extent. It is placed between the left trefoil of the central tendon anteriorly, and the muscular portion posteriorly and laterally (Fig. 391). The aperture is rounded or oval with its long axis placed trans-

versely, and there is usually no hernial sac. The stomach and a portion of the colon are invariably herniated, but the spleen, portions of small

intestine, and occasionally the greater part of the large intestine, may be intrathoracic. The position of the defect corresponds almost exactly with the level of the entrance of the phrenic nerves into the diaphragm, and therefore with the point of junction of the anterior half developed from the septum and the postero-lateral portion developed from the pleuro-peritoneal folds. One may, however, study embryological reconstructions in vain for a possible stage in which the pleuro-peritoneal folds do not meet the septum, and in which mobile intestine is available for herniation, but one is forced to the conclusion that normally no opening is present in this region. There are two possible explanations of the defect. The first is that there is delay or interference with the growth of the pleuro-peritoneal folds, and that in consequence they fail to unite with the dorsal portion of the septum transversum. The second is that herniation occurs after the membranous diaphragm has formed, resulting in either a true (or sacculated) or a false (or non-sacculated) hernia.

To consider the first of these in more detail, it is possible that the growth of the pleuro-peritoneal folds might not progress as rapidly as it should in the region of the septum. In order to account for this it is necessary to realize what factors could influence the growth of the diaphragmatic primordium, especially in this region. Dissociation of growth of various tissues has been experimentally produced by the alteration of the nutrition of embryonal cells, and various teratological abnormalities such as monsters, hare-lip, spina bifida, etc., have been shown to be due to this cause. It is therefore possible that some temporary disturbance of nutrition might affect the relative rate of growth of the components of the diaphragmatic primordium.

There is little doubt that the growth of the pleuro-peritoneal folds is dependent on the growth of the liver, to which organ they act as suspensory ligaments. Should delay in the growth of the liver occur, a defect might be produced anteriorly, but even this would be at a stage when the pleuro-peritoneal passage was still open and equally prepared to receive herniated structures. In fact, if this were the case, there is no apparent reason why a double hernia, through the dome and through the passage simultaneously, should not occur. From consideration of the varieties of hernia through the pleuro-peritoneal passage, it is apparent that when there is markedly defective growth of the pleuro-peritoneal folds a very large opening remains, extending right into the dome or even more anteriorly.

The approximate time at which herniation occurs through the pleuro-peritoneal passage has already been considered—namely, at the time of the return of the intestine from the umbilical stalk—and this must be the same in the case of hernia through the dome.

The second possibility, the occurrence of herniation after the membranous diaphragm has formed, is much more likely. At this time the pleural cavity is relatively unfilled by the developing lung bud, and a portion of membranous diaphragm lies unprotected, at any rate on the left side, owing to the inequality in size of the liver lobes at this stage.

In the acquired types of hernia the important factor is a momentary sudden rise of intra-abdominal pressure. If evidence could be produced of the occurrence of this in the embryo at the time when the diaphragm is largely membranous, the theory might be placed upon a more substantial basis.

What factors might produce an increase of intra-abdominal pressure in the embryo? It has been suggested by Gruber that the development of the rectus abdominis, the pressure of the flexed lower limbs, flexion of the caudal region, or even growth of the liver, may cause it. None of these seems very probable, nor do oligamnios and hydramnios, which are so frequently suggested as causes for any congenital abnormality.

In a 20- to 30-mm. embryo—that is, up to the seventh week—the abdominal cavity is small, and the intestines are lying in the umbilical stalk, the neck of which is partially obstructed by the edge of the liver. When the embryo has reached about 40 mm. the intestine suddenly leaves the umbilical stalk and enters the abdominal cavity. This is due to two factors, first, an increase in the size of the abdomen accompanied by a relative diminution in the size of the liver, and, second, a disproportion between the rate of growth of the intestine and the umbilical stalk. A moment arrives when the intra-abdominal pressure is lower than the pressure in the umbilical stalk, so that the contents of the latter slip into the abdomen. At the moment of return the intra-abdominal pressure must be increased. Obviously the time at which this takes place must vary, and it is in those cases in which the intestine returns earlier than usual, while the diaphragm is still membranous, that herniation may occur, a portion of the membrane giving way and intestine passing into the pleural cavity uncovered by a hernial sac. In a hernia produced in this way the subsequent development of the diaphragmatic musculature would give a firm margin to the ring so that it would have a rounded, punched-out appearance.

Further support of this view may be found in cases recorded by Beckman, Duval, and the present writer in which the hernia was associated with absence of rotation of the gut. The large intestine lay to the left and partly in front of the small intestine, in the position occupied before the normal axial rotation of the gut is completed. It is therefore probable that the intestine passed through the diaphragm at or soon after its return to the abdomen.

A partially detached lobe of the liver is a not infrequent content of herniæ through the dome, several cases being recorded by Keith, and others by Monks, Ovi, and Devulder. In such cases the penetration must have been produced by the abnormal growth of such a lobe, and practically all herniæ through the right dome are associated with abnormal development of the right lobe of the liver.

It has been suggested that pressure during the third month of intra-uterine life may prevent the invasion of the membranous diaphragm by muscle, and so allow a sacculated hernia to be formed, but it seems more probable that sacculated herniæ in the dome are acquired.

### 3. HERNIA THROUGH THE ŒSOPHAGEAL ORIFICE.—

*a. Thoracic Stomach.*—The first two cases of this condition on record were described by Bailey and by the present writer, and since then at least six others have been published. Varying portions of the fundus and body of the stomach lie in a peritoneal sac in the posterior mediastinum, and the Œsophageal orifice is in consequence enlarged to accommodate the stomach which passes through it, though some degree of constriction is usually present. The greater and lesser omenta extend upwards along the curvatures, carrying

in their folds the coronary and epiploic vessels, which have a normal origin. The cardiac orifice is intrathoracic, and may be as high as the level of the root of the lung.

The condition can hardly be considered as a hernia, but is due to the failure of the caudal migration of the stomach to keep pace with the descent of the other organs. In a 4.5-mm. embryo the stomach lies in the lower cervical region, separated from the heart by the septum transversum. It gradually descends from this position, though in opposition to, rather than in company with, the septum transversum, as stages can be seen in which it lies cranial to the dorsal margin of the septum. The cranial position of the stomach in relation to the septum transversum always leaves a record behind it, for a portion of the lesser sac persists for a time in the posterior mediastinum as the infracardiac bursa of Broman.

This caudal migration of the stomach is brought about by the growth of the œsophagus, and by the straightening out of the cranial flexure of the embryo. In the human embryo the migration is almost complete before the final constitution of the diaphragm. When the tailward migration fails to take place, the stomach, instead of the œsophagus, is found in the position of the normal œsophageal opening in the diaphragm.

The upper part of the cœlomic cavity, formed by a splitting of the mesoderm in that region, surrounds the stomach, and provides in cases of thoracic stomach a peritoneal sac in the posterior mediastinum to accommodate it.

*b. Para-œsophageal Hernia.*—This is a fairly common variety of hernia occurring through the œsophageal orifice. A protrusion of the upper part

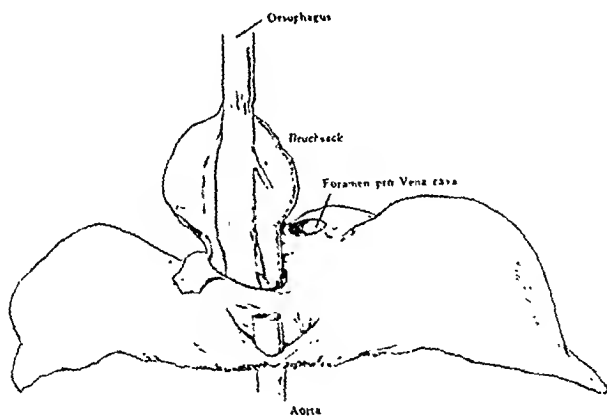


FIG. 392.—A diaphragm showing a para-œsophageal hernia. (Gruber.)

of the lesser sac of the peritoneum (omental bursa) passes through the orifice to the right of the œsophagus, and extends upwards into the posterior mediastinum. The sac varies in size from a small finger-like process to a dilated sac of considerable dimensions. The œsophagus is probably always of normal length, and the orifice is slightly enlarged. (Fig. 392.)

In the absence of adequate cause such as trauma or inflammation, it is difficult to believe that a normal diaphragm with properly developed crura

could ever be the seat of a para-oesophageal hernia. Nevertheless, the discovery of such herniæ in mature and elderly subjects has led to controversy about the possibility of their being acquired. I believe that such herniæ always start from a congenital defect in the diaphragm, the occupation of such a defect by hernial contents being an event which may occur by chance at almost any epoch in life.

A defect in this region must arise in connection with the incorporation of the tissues of the dorsal mesentery with the dorsal and median or the crural portion of the diaphragm. This element in the constitution of the diaphragm is, of course, in being before any other of the diaphragmatic members, and is the seat of the formation of the lesser sac or omental bursa, which begins in all vertebrates as a slit-like space along the right margin of the mesentery. This slit extends cranially as far as the root of the lung. When the diaphragm is completed by muscular invasion during the second and third months of intra-uterine life this cranial extension becomes isolated and usually disappears, though occasionally it may persist.

If the muscular invasion of the diaphragm were incomplete here, then the cranial extension of the lesser sac could remain in connection with the peritoneal cavity of the abdomen, or conversely its persistence would retard and hinder the muscular invasion. If the stomach herniated into it, there would be formed a para-oesophageal hernia passing into the posterior mediastinum to the right of the oesophagus. The hernial sac would be formed by the remains of the cranial portion of the lesser sac.

The expectations roused by such a view of the origin of para-oesophageal hernia are almost perfectly realized. The herniæ do occur on the right side of the oesophagus, and they are covered by a membrane whose margins are continuous with the lesser sac of the peritoneum, and such pre-formed sacs are found quite frequently in the dissection of fetuses.

A survey of recorded cases convinces us that this interpretation is sufficient to account for the majority of para-oesophageal herniæ. In the cases of mature or elderly subjects, where most doubt as to their congenital origin would naturally arise, careful dissection has corroborated the congenital view of their origin. For in these cases evidence of a membranous sac is usually forthcoming, though, as is natural enough, it may not be always complete. Always the peritoneum covering the stomach, the usual content of this kind of hernia, comes away completely from the membranous sac. There is no fusion between them. On the other hand, bands of muscle and fibrous tissue often pass from the oesophagus to the membranous lining, suggestive of a congenital relation between the two.

### ACQUIRED DIAPHRAGMATIC HERNIA.

1. **Non-traumatic.**—The causation of acquired diaphragmatic hernia has long been summed up in the single word 'trauma'. It is obvious that penetrating wounds of the diaphragm, whether inflicted by the ends of fractured ribs or by implements of violence, will produce an opening through which herniation could occur. There is, however, a place in the pathology of acquired diaphragmatic hernia for a non-traumatic group.



In the absence of any pre-formed congenital sac or congenital defect there are two possible methods by which herniation may take place. These are, first, the gradual protrusion of a hernial sac through a relatively weak spot in the normally developed diaphragm, and, second, the production of an acquired defect owing to the rupture of some of the muscle fibres at their point of insertion into the central tendon.

Intra-abdominal pressure plays a very important part in the actual production of herniation, by forcing the abdominal contents either into a pre-formed congenital sac, or, with a covering of peritoneum, through a defect in the parietes. During violent muscular effort the intra-abdominal pressure may rise to 100 or 150 mm. of Hg, due partly to the descent of the diaphragm but mainly to the contraction of the anterior and lateral muscles of the abdominal wall, especially the transversalis abdominis. When the intra-abdominal pressure is raised the diaphragm is in close relation to the structures passing through it, which greatly reduces the possibility of herniation through one of the normal diaphragmatic apertures. In spite of this fact a few extremely rare forms of diaphragmatic hernia are probably due entirely to the direct action of intra-abdominal pressure. These are herniæ through the foramen of Morgagni, or para-oesophageal herniæ, anterior or to the left of the oesophagus.

On the other hand, a number of diaphragmatic herniæ occurring in the dome of the diaphragm are not recognized, nor do they produce symptoms until the fourth or fifth decades. It is conceivable that such a hernia, if small and covered by a sac, might be present from birth and not produce symptoms, though radiologists have found numerous cases accounting for indefinite abdominal symptoms that had defied diagnosis by other means. Such cases must be excluded from the congenital group, and as there is no structure passing through the diaphragm in this situation they can only be due to some pathological process.

A muscle may be ruptured by the force of a sudden strain during contraction, although the rupture need not depend on direct injury to the muscle. Rupture almost invariably occurs at the point of junction of muscle fibres with aponeurosis, or within an inch of that point. The complete rupture of such a muscle as the rectus femoris is a well-known example, but those who are called upon to remedy comparatively trivial but yet disabling injuries in athletes are familiar with the fact that the tearing of a few muscle fibres is a commonplace occurrence, especially in those who are 'out of training' or attempt violent exertion without any form of preparation.

It is impossible to produce direct evidence that the muscular fibres of the diaphragm are torn from the central tendon when the intra-abdominal pressure is suddenly raised. A little consideration, however, will show that the arrangement of the diaphragm makes such an occurrence possible. The muscle fibres of the diaphragm are spread out in a thin sheet, running an average length of 12 cm. from their origin to their insertion. They are adapted to performing some 20 to 40 contractions per minute against an average intra-abdominal pressure of 60 mm. of Hg. In the porpoise the tendon is greatly reduced in size, and the muscle is both increased in length and thickened

owing to the rapidity of the inspiratory movements required when the animal reaches the surface. The muscle is not only relatively but actually thicker in the porpoise than in man.

In man at the moment of forced inspiration the muscular fibres of the diaphragm have contracted to their fullest extent. It has been shown by Halls Dally, using the orthodiagram, that the distance between the central tendon and the point of origin in the lateral plane is 15.6 cm. in full expiration and 13.2 cm. in full inspiration, a contraction of 2.4 cm. While this takes place the domes of the diaphragm descend, the right 3.4 cm. and the left 3.2 cm.

The maximum intra-abdominal pressure that can be produced by violent muscular effort is about 150 mm. of Hg. Such pressure may be regarded as a fluid force pressing equally against the parietes and at right angles to them (*Fig. 393*). In the case of the

diaphragm the muscle fibres are in the main supported by the thoracic framework in the region of the phrenico-costal recess, but the region of the dome is unsupported, and in the case of the left side entirely unprotected. It will be at once seen that the weakest point is situated at the junction of the central tendon and the muscle, and this area of junction is exactly where rupture occurs in other muscles. Hence, supposing that in the fourth or fifth decade of life, a diaphragm which had been weakened by not being called upon to contract to its fullest extent, and which perhaps was also weakened by some degree of fatty infiltration, was suddenly called upon to withstand a pressure of 150 mm. of Hg, it is quite possible that

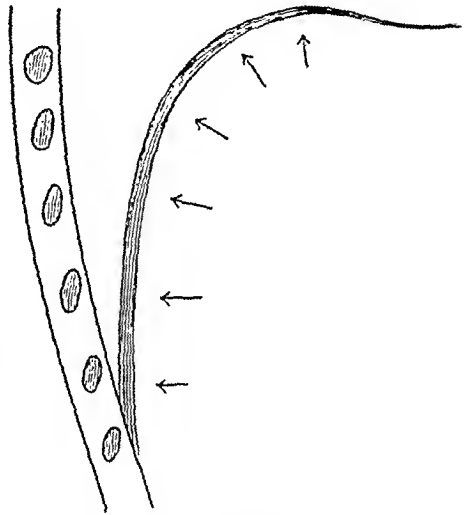


FIG. 393.—Diagram showing the direction of the intra-abdominal pressure acting upon the diaphragm.

some of the fibres might rupture and become detached from the central tendon. Such a rupture would not affect either the peritoneal or the pleural covering, but the muscle fibres would retract for about 2.5 to 3 cm., leaving an oval defect with its long axis in the line of the muscle fibres. The retracted ends would curl up and become covered with blood-clot, so that the edge would ultimately become smooth, rounded, and firm. Once such a defect had been produced in the musculature, it would only be a matter of time for the protrusion into the pleural cavity of a sac lined by peritoneum and covered by pleura, into which any of the mobile contents of the upper abdomen might pass.

As has already been indicated, a congenital hernia through the dome is more likely to be without a hernial sac, while if this theory is accepted it will be seen that an acquired hernia in the same situation will be sacculated.

**EVENTRATION.**—This is an elevation or generalized bulging of one dome, but it cannot properly be classed as a hernia. It occurs as a result of

unilateral phrenic paralysis, either following phrenic excresis or post-diphtheritic paralysis, or temporarily as a result of subdiaphragmatic inflammation. The diaphragm may also be displaced upwards by the pressure of an abscess or tumour.

A congenital form has been described in which the dome consists of only a thin fibrous membrane, and it has been ascribed to failure of development of the muscular elements of the diaphragm, or even of the phrenic nerve. The latter defect is highly improbable, but failure of muscular invasion might occur if the diaphragmatic primordium were subjected to pressure.

**2. Traumatic.**—The subject of injuries of the diaphragm was so exhaustively discussed by Gordon Bryan in his Hunterian Lecture in 1921 that it is out of place here to make more than passing reference to the subject. He showed that in severe crushing injuries the diaphragm may be ruptured by stretching and bursting mechanism, or torn by the sharp end of a fractured rib, a missile, or a weapon, by the penetration of a fractured a traumatic diaphragmatic hernia, which in the great majority of cases will be devoid of a hernial sac. The rapidity of the development of symptoms will naturally depend on the degree of injury to the diaphragm and the ease with which the abdominal viscera can be forced into the pleural cavity. Occasionally small wounds of the diaphragm heal spontaneously, especially on the right side. On the left side they may become sealed by adhesions. If a hernia develops, adhesions are almost always present at the margin of the opening, and in some cases the opening may be very difficult to define on this account. Frequently the herniated structures are adherent to each other and to the thoracic contents.

In conclusion I should like to express my thanks to the President and Council of the College for conferring on me the privilege of delivering this lecture, and to my friends Professor Woollard and Professor J. E. Frazer for the help they have given me in its preparation.

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## THE TREATMENT OF DUPUYTREN'S CONTRACTURE.

### A REVIEW OF 31 CASES, WITH AN ASSESSMENT OF THE COMPARATIVE VALUE OF DIFFERENT METHODS OF TREATMENT.

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THE present paper embodies the results of an inquiry undertaken by the writer when Surgical Registrar at the Manchester Royal Infirmary. Although the number of cases may appear somewhat small, it represents the largest series of end-results recorded in the literature of this condition.

In spite of the fairly common occurrence of the lesion, the patients come but seldom to operation, no doubt on account of the fact that few find the disease sufficiently inconvenient to necessitate surgical relief. A further explanation probably lies in the reluctance of many practitioners to advise a treatment which experience has shown them to be uncertain—to say the least—in its promise of a permanent cure. That such a view is not unjustified must be admitted, but it has had the unfortunate effect of delaying operative procedure until contraction is extreme, making impossible complete eradication of diseased tissue, and permitting the secondary changes in skin and joints which are the so frequent cause of recurrence.

Each of the 31 cases has been personally seen and examined, and it is suggested that the results so obtained may help somewhat in the formation of a rather more reliable index of prognosis than has hitherto been obtainable.

### ETIOLOGY.

The cause of the contracture continues to remain obscure in spite of the multiplicity of theories which have been advanced since Dupuytren<sup>1</sup> first ascribed it to chronic irritation. Loewy<sup>2,3</sup> considers it the result of an "hereditary tendency to fibrous hyperplasia" and records its appearance in four successive generations of one family, while Robert Jones<sup>4</sup> would combine "chronic palmar irritation with an individual predisposition". Wainwright<sup>5</sup> suggests an "endocrine deficiency", Schubert<sup>6</sup> a "neuropathic influence secondary to ulnar nerve fibrosis", Ely<sup>7</sup> an inflammatory fibrosis the result of diseased tonsils, and Byford<sup>8</sup> a similar reaction due to the presence of dental sepsis. Adams<sup>9</sup> favours a "constitutional cause", while Teschemacher's<sup>10</sup> list of 213 cases included 33 with associated diabetes mellitus. Lead poisoning is ascribed as a cause by Michaux, Lemaître, and Picard<sup>11</sup>; Krogius<sup>12</sup> considers it a developmental disease due to disorders of growth in the superficial palmar muscles; Tubby<sup>13</sup> "a fibrositis, or a local expression of some other change in bodily metabolism". A single definite

interphalangeal joints; and good results are claimed following excision of the involved tissue.

The uniformly good results following multiple subcutaneous fasciotomy will be referred to later.

In considering the question of treatment in general, a review of the literature reveals an almost bewildering variety of different methods. Some attempt will be made to correlate their results, in an endeavour to estimate their relative values, but perhaps the more obsolete may be eliminated as a preliminary. Massage, manipulation, extension, hot-water baths, passive hyperæmia, ionic medication, and counter-irritation have been tried for generations without effect, although occasional successes have been claimed following their employment. A case recently reported by Trumper<sup>15</sup> appears to have been much relieved by counter-irritation with iodine. He produced a bullous dermatitis by the inunction of the unguentum iodi denegrescens daily for ten days in a case of palmar fascial contracture following an injury, with resulting apparent cure. The permanency of the effect would seem open to considerable doubt, and the fact that the original condition was not a true Dupuytren's contracture somewhat minimizes the value of the observation. It would, in addition, appear doubtful whether heroic measures of this type applied to the susceptible skin of the palm are justified by the results published to date.

A similar remark would apply to those methods which depend upon the introduction of some foreign body, such as fibrolysin, into the subcutaneous palmar tissues; yet Stahlke<sup>16</sup> claims good results in 12 cases following the injection of sterilized human fat, and it must be admitted that a certain amount of difficulty is encountered in interpreting this result. The thickening remains, but the presence of the fat "appears to mobilize the fingers". Acting on the assumption that the disease is a manifestation of an endocrine disorder Leopold-Levi<sup>17</sup> and Wainwright<sup>5</sup> have used thyroid extract internally, with some degree of success. The former treated 7 cases with 10 cgm. of thyroideum siccum daily, using up to 150 doses in one case. He states 5 of the patients were "somewhat improved". Wainwright, on the other hand, claims "excellent" results in 4 cases treated over a period of years with  $\frac{1}{2}$  gr. of the extract daily. This incompatibility remains unexplained, but it is questionable whether the advisability of the prolonged administration of even small doses of the gland in the absence of definite signs of thyroid deficiency is sustained.

But apart from treatment by radium, which will be considered later, operation of some type remains the method of choice, though Keen's<sup>18</sup> direction that "the rational measure is the excision of the contracted fascia" is open to question. Dupuytren<sup>1</sup> himself used to make one or more transverse incisions, stretch the fingers straight, and allow the wound to heal by granulation, and the modern fasciotomy is the same method scientifically applied. Multiple subcutaneous fasciotomy was described by Adams<sup>9</sup> in 1890, and it remains to-day an operation of considerable value, perhaps of greatest value, in many cases of Dupuytren's contracture. Adams used a special knife, but any kind of fine sharp tenotomy knife is suitable. The instrument is inserted between the skin and tense band, and the latter carefully divided. From five to fifteen incisions may be needed, according to the severity of the cases.

Continuous splinting for a week is adopted, followed by massage and manipulation with night splint only for a further month. It is an exceedingly simple operation, requires local anæsthesia only for its performance, necessitates no in-patient treatment, leaves no residue of anæsthesia or sear neurosis, can be repeated at intervals if necessary, and gives excellent immediate results. As against these advantages, it is purely a palliative expedient, and, in addition, unless the knife is used extremely carefully, might result in injury to tendon-sheath, vessels, or nerves. Its efficacy is well illustrated in the above series, in which 6 hands remained free from recurrence from one to five years after operation, while 1 showed partial and 1 complete return at intervals of two and four years respectively.

The unsatisfactory sequelæ and tendency to recurrence following the operation of fascial excision and suture has led to many modifications in procedure. Variations in the type of incision are numerous. Hutchinson<sup>14</sup> deprecates the use of the V and Y incisions, on account of the excessive scarring of the palm which remains. He advocates the usual longitudinal incision. Russ<sup>19</sup> employs incisions 1 cm. in length along the fingers over the affected joints, and through them excises the underlying fibrous bands, dividing their lateral expansions after identifying them with the knife point. The palmar bands and nodules are excised through similar small incisions placed appropriately. Gill<sup>20, 21</sup> insists on "the value of incisions along the natural creases of the hand and fingers in order to secure healing of the wound without danger of subsequent keloid growth, contracture, and adhesion of the sear to underlying structures." He finds it possible to remove all the palmar fascia involved through a transverse incision along the distal palmar crease, and its extensions through transverse incisions along the crease at the base of the fingers. Where skin-grafting is contemplated the Y is the incision of choice; when immediate skin union is intended it appears uncertain that small multiple incisions will allow of complete fascial excision. In these cases the choice would appear to lie between the original longitudinal and Gill's transverse incisions, though one suspects that the section of so many superficial vessels during the latter procedure must seriously impair the nutrition of skin already destined to be separated from its deeper vascular supply. It is probable that the older method still remains to be preferred.

In those cases where excision of affected skin has made it impossible to draw the edge together without tension, some type of skin-graft is required. Kilner<sup>22</sup> employs Thiersch grafts; Kanavel,<sup>23, 24</sup> Davis,<sup>25</sup> and Koch<sup>26</sup> prefer a free full-thickness skin-graft; while the pedicle graft has been occasionally used. The free whole-skin graft would appear to be the most useful. It is, paradoxically enough, more certain than the pedicle graft, which has a tendency to slough when applied to this situation (Kanavel<sup>23</sup>), and dispenses with the prolonged fixation and additional operation consequent on the latter procedure. Although not so cosmetically desirable as Thiersch grafting, it is the method of choice in working men, in whom the thinner grafts are too delicate to withstand the heavy wear and tear to which they are subjected. In the case of women, and in men of the professional and leisured classes, the reverse opinion would apply more correctly. Where the little finger is hopelessly contracted, and the palmar skin much involved, Chitty, of Bristol,

'fillets' this digit, using the residue for skin-grafting the excised integument. A similar procedure is adopted by Wrigley, of Manchester.

The question of sueutaneons tissue transplantation to replace the excised fascia has been much debated. Peiser<sup>27</sup> reports success following the subcutaneous insertion of a free fat-graft in Dupuytren's contracture, and Spitzzy<sup>28</sup> has used similar grafts in other conditions of the palm with good results. Gill,<sup>20</sup> acting upon similar experience which "has proved to him the great value of the free fat transplant in preventing adhesions after excision of scar tissue and after extensive dissections", discusses a case of Dupuytren's disease treated in this way, with apparently good result. But it should be noted that the wound rapidly broke down at one end, and discharged serum and liquid fat for weeks. A similar sequel occurred in a case reported by Kanavel and others.<sup>24</sup> Abbott<sup>29</sup> replaced the excised fascia by a sheet of fascia lata, cut to size (allowing for shrinkage) and anchored with a few fine catgut stitches round its edge. He reports one case showing an excellent result two years after operation.

A consideration of methods involving subcutaneous fat or fascial implantation cannot but give rise to a certain amount of doubt concerning their field of usefulness in the conditions under notice. Such procedures are only possible in those cases where the skin edges can easily be drawn together—that is, in cases which would almost certainly be cured without their employment. The risk of sepsis consequent on the introduction of a badly vascularized foreign body is illustrated in the two cases quoted above. In addition, an extremely long convalescence is required, and in the case of fascial implantation a degree of skin undercutting necessitated which must severely jeopardize the vascularity of the integument. It remains to be seen, however, whether these purely theoretical objections are justified.

Two further operative modifications require notice. Hutchinson,<sup>14</sup> believing recurrences to be the result of residual secondary changes, advocates the excision of the head of the proximal phalanx and shortening of the extensor tendon through a small dorsal incision. He obtained excellent results following this procedure. Tubby,<sup>13</sup> following fascial excision in the usual way, rubs fibrolysin into the wound in addition to injecting it in the surrounding tissue. He considers that this measure prevents the painful wire-like scar which occasionally results, and claims a cure-rate of 90 per cent in his cases. It is doubtful, however, whether these results are *post hoc* or *propter hoc*.

An excellent commentary on the various operative procedures discussed above is provided by a consideration of the results in 29 cases of Dupuytren's contracture reported by Kanavel, Koeh, and Mason.<sup>24</sup> In the majority of the cases fascial excision with skin suture was practised, while others (too few to be of statistical value) were treated additionally by full-thickness grafts, free fatty grafts, pedicle grafts, or by Hutchinson's operation. Fascial excision was performed extremely carefully, stress being laid on "the importance of excision not only of the contracted fascia, but of all its attachments to the skin, the interfascial septa, the interosseous fascia, the metacarpal bone and the phalanges." "We believe", they state, "that the essential factors in the treatment are as complete an excision of the palmar fascia as can be accomplished through the operative incision most suitable for the case in

question, the excision of hopelessly affected skin, and primary closure of the wound without undue tension. In some cases this may involve the use of a free full-thickness graft of skin to replace the excised covering tissue." Of the 29 cases, 20 (roughly 70 per cent) were considered "good" results. In 5 (17 per cent) the results were "fair", while in 2 cases (7 per cent) they were "definitely unsatisfactory". Guarantee of cure, it will be observed, remains far from certain, even following major operative procedures.

**Radium and X-ray Treatment.**—Radium has been used sporadically for the relief of Dupuytren's contracture, with very varying results. Apert<sup>30</sup> records a case in which some relaxation of the fingers followed exposure, allowing the patient free use of the hand although the palmar thickening remained. Specklin and Stoeber<sup>31</sup> obtained better results in a case in which both plantar and palmar fasciæ were contracted, with symptoms of peripheral neuritis. Of 5 cases reported from Stockholm in 1929, 3 abandoned treatment, 1 was not improved at all, and the fifth was somewhat better. In a case seen by the writer recently, the only result of the application of a radium plaque is an intractable and extremely painful burn, the contracture being unaffected.

It would appear from these few reported cases that radium as a therapeutic agent in this connection is of extremely doubtful value, and might on occasion be worse than useless. But theoretical considerations incline one to the view that further experiment might reverse this judgement. The rapid softening of the ordinary keloid scar following surface application is well known, and it is suggested that experimental dosage might exercise a similar effect upon the subcutaneous fibrosis of Dupuytren's contracture. "The amount of radium used in softening keloids is not unlike the 'carcinoma dose', and it would seem that as malignant tissue is more radio-sensitive than normal, and requires about the same dosage for cure as keloid does for absorption, that the action in the latter case is in the nature of a stimulative phagocytosis."<sup>32</sup> However, it must be admitted that radium must remain little more than a factor of promise until further experiment proves its value.

X-ray treatment has occasionally been advocated, but Wigoder<sup>33</sup> found fibrous tissue experimentally to be radio-resistant to X rays, and, in the testis, doses which caused all the sperm line to disappear had no similar effect on the supporting cells, which are more or less like fibrous cells. In fact, they overgrew, probably on account of the extra space provided for their expansion. The fibrosis consequent upon the treatment of exophthalmic goitre by X rays is also well known. It is conceivable that the employment of X rays in the treatment of the contracture might have an effect the reverse of improvement, and their use should be recommended with considerable caution.

### CONCLUSIONS.

1. The etiology of Dupuytren's contracture remains obscure.
2. Mechanical methods of treatment, including fibrolysin and radium and X-ray therapy, are useless, with the proviso that further experience may conceivably make radium a safe and effective cure.
3. Operation of some type remains the method of choice in treatment. It is suggested that as the results from even the most major operative



procedures are far from satisfactory, the minor operation of multiple subcutaneous fascial division should be adopted, and repeated if necessary, in the average patient of the hospital class. In patients of the more leisured class, wide fascial excision with replacement of involved skin by a free full-thickness skin-graft is probably most advisable, combined, if necessary, with Hutchinson's operation. Where the best cosmetic result is desirable, e.g., in non-labouring women and in men of the professional classes, Thiersch grafts may be substituted for the thicker full-thickness one. Implants of fat or fascia are theoretically undesirable, and have not been uniformly successful in application.

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# DUPUYTREN'S CONTRACTURE

547

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## URETEROCELE:

### SOME OBSERVATIONS BASED ON THE INVESTIGATION AND TREATMENT OF FOUR CASES.

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A URETEROCELE is a cystic swelling of the intramural part of a ureter due to prolapse of one or more of its coats, projecting into the bladder and invariably associated with a pin-hole ureteric orifice. This abnormal contraction of the aperture, which may be congenital or acquired, leads to the formation of a ureteroceles in the following way.

In a normal subject the pelvis of the kidney becomes gradually distended with urine until its tension is sufficient to bring about a peristaltic wave of the musculature of the upper urinary tract, thus squeezing the liquid down the ureter to its lower part, whence it passes into the bladder by a characteristic gush through the ureteric orifice. As seen through the cystoscope this gush is observed to last for about two seconds, and to be accompanied by slight dilatation of the intramural part of the ureter, showing that the opening offers some resistance to the passage of urine through it. In cases of pin-hole orifice this resistance is greatly increased, the duration of the outflow much prolonged, and the swelling of the intramural part of the ureter markedly accentuated. Such partial obstruction brings about a detachment and stretching of the mucous membrane at the vesico-ureteral junction, culminating in the formation of a ureteroceles.

The condition is said to be bilateral in about 10 per cent of cases,<sup>1</sup> and to be more common in the female sex. Of the four cases described in this paper, one was bilateral, and three of the patients were women.

**Varieties of Ureteroceles and Cystoscopic Appearances.**—Ureteroceles are divided into two varieties which may be called, respectively, mucous and muscular.

1. *Mucous Type.*—In this type the cyst wall is composed of a double fold of mucous membrane only, the outer being vesical and the inner ureteral in origin, both transitional in type. This variety, which is the more common (three cases), appears translucent when seen through the cystoscope, with clearly defined blood-vessels radiating over its surface.

2. *Muscular Type.*—In this type the inner and outer walls consist of mucous membrane as before, but interposed between these is a varying amount of muscular tissue derived from the wall of the ureter (one case). The cyst is opaque, while the blood-vessels are fewer in number and less clearly defined.

If in either kind of ureteroceles the ureteric orifice is watched, the cyst will be seen to swell up suddenly, owing to distension of its lumen by a

peristaltic rush of urine. Next follows a small prolonged efflux through the contracted opening with gradual shrinking in size of the cyst, which presents a convoluted worm-like appearance when completely emptied. With a further rush of urine from the kidney the cycle recommences.

These changes can only be observed when the bladder is moderately distended with urine. Should too much fluid be present, the intravesical pressure becomes greater than the intra-ureteric, and for obvious reasons the cyst is unable to expand. On allowing fluid to escape gradually, the cyst will soon be seen to expand, the degree increasing with each efflux as the surrounding pressure diminishes.

The orifice, as a rule, is situated at the centre of a small nipple or papilla, which does not take part in the general expansion, and is single (three cases). This projection may be at the summit of the cyst, when it is readily seen, or laterally placed, when, if posterior, it only comes into view when the ureterocele is collapsed. In one case no such nipple was seen, and the orifice was only observed after much searching; when collapsed a second opening was found at the junction of the cyst and the intramural ureter. This double orifice has to my knowledge never been observed before, and may therefore be regarded as unique (*Case 3*).

**Symptoms.**—These are of two kinds, upper and lower urinary tract symptoms.

1. **THE UPPER-TRACT SYMPTOMS.**—These are referable to the mechanical effects of the contracted orifice resulting in back-pressure on the ureter and kidney with varying degrees of hydronephrosis and hydro-ureter. Infection may become superadded, and pyelonephritis, pyonephrosis, and stone formation follow.

*a. Renal Pain.*—This is usually of a dull, aching character, located in one or other loin (all four cases). Occasionally definite attacks of renal colic may occur, but only in presence of infection with stone formation.

*b. Ureteric Pain.*—This may vary from slight prickling sensations in the groin to definite, but mild, attacks of ureteric colic due to irregular contractions of the ureter, by which attempts are made to force the contained urine through the ureteric orifice.

*c. Hæmaturia* (three cases).—This may be the only symptom (one case), in all probability of renal origin and due to congestion. In cases complicated by stone formation it may result from trauma.

2. **THE LOWER-TRACT SYMPTOMS.**—These are due to the presence of the ureterocele in the bladder with or without infection.

*a. Vesical Irritation.*—This may cause frequency of micturition (two cases), and often the patient feels that there is residual urine that cannot be passed (one case). If infection is present, this in itself may cause frequency.

*b. Urethral Obstruction.*—Sometimes with a large cyst there may be actual obstruction to the passage of urine from the bladder through the internal meatus, and occasionally retention.

*c. In females* the cyst has been reported to have appeared at the external meatus and to be visible on inspection.

**Diagnosis.**—This depends for its recognition on the use of the cystoscope, for there is no symptom, or group of symptoms, which cause its

presence to be suspected. Renal aching, hæmaturia, and frequency, call for a complete urological investigation, and this will, of course, make the diagnosis clear.

Ureteric catheterization is impossible owing to the smallness of the ureteric orifice, and should be replaced by the intravenous injection of abrodin. This will indicate the presence of hydronephrosis and any complications such as stone.

**Treatment.**—This may be of two kinds, suprapubic or per-urethral.

1. SUPRAPUBIC TREATMENT consists of opening the bladder as for the first stage of a prostatectomy, and ranges from the simple slitting of the cyst to complete resection with or without circular suture of the two mucous layers.<sup>2</sup> It was not employed in any of the cases. In the presence of gross complications, such as large hydronephrosis, hydro-ureter, or extensive inflammatory changes, nephrectomy or nephro-ureterectomy may be indicated.

2. PER-URETHRAL TREATMENT consists of the application of instruments or diathermic electrodes to the ureteroceles through a cystoscope. The instruments which have been employed are numerous, and include graduated sizes of ureteric bougies, cystoscopic scissors, Young's cystoscopic rongeur, etc. The diathermic electrodes are of two kinds: (a) insulated snare, and (b) simple cystoscopic fulguration electrodes.

a. *The Insulated Snare* as devised by Foley<sup>5</sup> consists of a length of fine wire passed double through a segment of a F.5 ureteric catheter with the loop projecting at one end. The catheter is threaded through a length of F.10 rubber catheter to provide the necessary insulation. A light mineral oil is used to distend the bladder to prevent dissemination of energy of the cutting diathermic current which is used. The ureterocoele can, by this electrode, be amputated and subsequently removed with cystoscopic forceps.

b. *The Simple Cystoscopic Electrode.*<sup>3</sup>—This was used in all four cases as follows:—

*Preliminary treatment.*—The patient is placed on hexamine before meals, and an acid mixture after, for a period of three days, in order to minimize the possibility of post-instrumental infection.

*Anæsthesia.*—In females no anæsthetic is required. In the one male case local injection of the urethra with a mixture of  $\frac{1}{2}$  per cent cocaine hydrochloride and  $\frac{1}{2}$  per cent sodium bicarbonate in four successive doses of 2 drachms each gave perfect anæsthesia.

*Technique.*—The patient is placed in the lithotomy position on a cystoscopic couch, and the parts, after being cleansed, are isolated by a lithotomy towel. The cystoscope is introduced and the bladder washed out and distended with not more than 8 oz. of oxycyanide of mercury 1-6000. The indifferent electrode consists of a leaden belt round the waist or leg, and the active electrode is introduced through the cystoscope. The ureterocoele is brought into the field of vision, the opening located, the electrode applied as far as possible from it, and coagulation begun. An attempt should be made to destroy the cyst wall completely so that urine may have a free exit into the bladder. This is by no means always possible, for the cyst soon tends to collapse from reflex inhibition of renal excretion, thus making the application of the electrode a matter of great difficulty. In no case should

coagulation extend to the papilla on which the orifice is situated, as occlusion will certainly take place, and in the event of failure in rupturing the cyst complete blockage of the ureter of the affected side occurs (one case). Should rupture of the cyst wall be impossible by diathermy and the papilla become included in the coagulation, a pair of scissors should be passed through an operating cystoscope and a small area of coagulated tissue excised. At the completion of treatment the bladder is washed out and the cystoscope withdrawn.

*After-treatment.*—The patient is placed on alkalis and Contréxeville water for three days, after which hexamine and acids are again given.

*Results of treatment and cystoscopic appearances.*—During the first seven to fourteen days after the treatment the cyst wall gradually sloughs away, leaving a much enlarged ureteric opening. Cystoscopy at a week shows much of this slough still adherent; at a fortnight it is almost entirely separated. At both stages there is some degree of reactionary inflammation, or it may be some bullous œdema surrounding the orifice. At the end of six weeks the latter appears almost normal except for a slight permanent enlargement.

The character of the efflux will, of course, be noticed to have undergone a marked alteration. Instead of a small prolonged stream issuing from the orifice, there is a strong gush of short duration, with practically no distension of the intramural part of the ureter.

All symptoms previously complained of by the patient, especially the dull aching in the renal angle, the colicky pains along the line of the ureter, and the hæmaturia will have cleared up.

## CASE REPORTS.

*Case 1.*—Female, age 42. Left renal aching for some years. Two bouts of hæmaturia and some frequency. Cystoscopic examination showed a left-sided ureterocele of mucous type. No urinary infection. X-ray negative to stone. Cyst wall destroyed by diathermy and ruptured at the time; ureteric orifice with its papilla untouched. No symptoms after treatment. Cystoscopic examination one month later showed no sign of cyst. Cure.

*Case 2.*—Female, age 58.<sup>4</sup> Attacks of dull aching pain in one or both loins for years. Some frequency and a feeling that the bladder was not empty after micturition. Six bouts of hæmaturia, each of short duration. Cystoscopic examination showed bilateral ureteroceles of mucous types. X-ray negative to stone. Urine free from infection. Cyst walls coagulated by diathermy. Right cyst perforated, but not left. Six hours after treatment great pain in the left loin, discomfort in the right. Twenty-four hours after treatment vomiting, pain, and tenderness in both loins; 3 oz. only of urine passed. Forty-eight hours after treatment pain worse; 6 oz. of urine passed altogether. Cystoscopic examination showed right ureterocele collapsed, left much distended and immobile, its orifice appearing to be blocked by reactionary swelling from the diathermy. Left kidney therefore totally obstructed and reflex anuria present on the right side. Knife passed through an operating cystoscope and plunged into left ureterocele. Gush of urine was followed by collapse of the cyst. During the next twenty-four hours 140 oz. of urine passed, and 60 oz. the following day. In the course of three hours the acute pain changed to a dull ache, lasting for three days. Cystoscopy at a fortnight revealed traces of coagulated tissue surrounding the much enlarged orifices. The effluxes were of good volume, short duration, and unstained. Cure.

*Case 3.*—Male, age 26. Left renal aching for six years. No hæmaturia; no frequency. Periods of depression and lassitude. Cystoscopic examination under cocaine anaesthesia showed a left-sided ureterocoele of muscular type; two orifices, no papillæ (*Figs. 394, 395*). Right ureteric orifice normal (*Fig. 396*). Urine normal.



FIG. 394.—*Case 3.* Left-sided ureterocoele: distended.

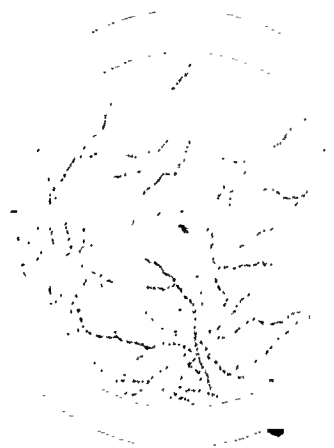


FIG. 395.—*Case 3.* Left-sided ureterocoele: collapsed.

X-ray negative. Cyst wall destroyed by diathermy. One orifice deliberately included in the coagulation, and drawings were made, as this shows how diathermy should not be applied where only one orifice is present (*Figs. 398–400*). Cyst not perforated. No symptoms after treatment because of second orifice,



FIG. 396.—*Case 3.* Normal right ureteric orifice.

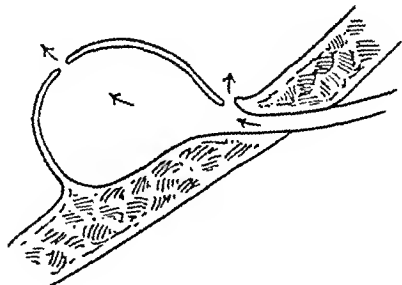


FIG. 397.—*Case 3.* Diagram showing double ureteric orifice. Occlusion of one orifice by diathermy consequently caused no obstruction.

and, consequently, no obstruction to the ureter (*Fig. 397*). Cystoscopy fourteen days later (*Fig. 401*) showed an excellent orifice; some surrounding bullous œdema. Cyst wall and some slough present. Final drawing not obtained owing to patient's departure abroad. Cure.



FIG. 398.—Case 3. Destruction of cyst wall by diathermy.



FIG. 399.—Case 3. Destruction of cyst wall by diathermy.



FIG. 400.—Case 3. Destruction of cyst wall by diathermy.



FIG. 401.—Case 3. Appearance fourteen days after operation.

*Case 4.*—Female, age 43. Attacks of painless hæmaturia for seven years. Some right renal aching at times. X-ray three years ago negative. Urine normal. Cystoscopic examination showed a right-sided ureterocele of mucous type with well-marked papilla centrally placed (*Figs. 402, 403*). Left ureteric orifice pin-hole, opening on summit of a papilla, but not small enough to have caused ureterocele (*Fig. 408*). Uroselectan: Both pelves and ureters normal. Diathermic terminal applied (*Figs. 404, 405*) and cyst wall coagulated (*Figs. 406, 407*) leaving nipple intact. Cyst wall not perforated. Some slight renal aching for three days. No diminution of urinary output. Some slight hæmaturia on the tenth to twelfth days. Nipple voided in urine on eleventh day and inspected. Cystoscopy three weeks after treatment (*Fig. 409*) showed remains of cyst wall with superimposed bullous œdema; orifice large and gaping. Cystoscopy six weeks later (*Fig. 410*)—orifice normal but much enlarged. Cure.





FIG. 402.—Case 4. Right-sided ureterocoele: distended.



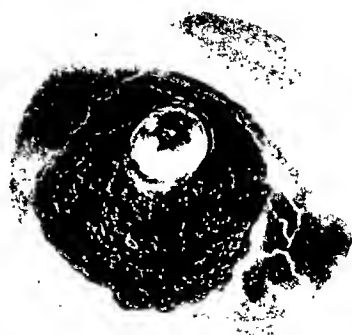
FIG. 403.—Case 4. Right-sided ureterocoele: collapsed.



FIG. 404.—Case 4. Application of diathermic terminal: right way.



FIG. 405.—Case 4. Application of diathermic terminal: wrong way.



FIGS. 406, 407.—Case 4. Coagulation of cyst wall.

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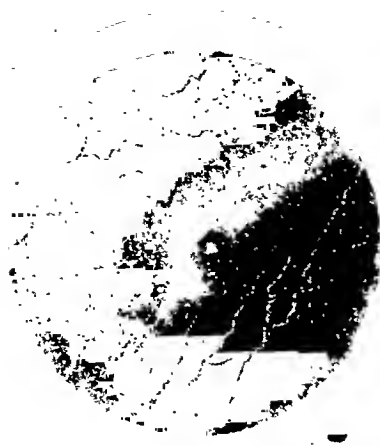


FIG. 408.—Case 4. Pin-hole ureteric orifice.



FIG. 409.—Case 4. Appearance three weeks after treatment.



FIG. 410.—Case 4. Appearance six weeks after treatment.

## SUMMARY.

1. Ureterocele is due to obstruction from a pin-hole ureteric orifice, which may be congenital or acquired, unilateral or bilateral.

2. Ureterocele, when diagnosed, should be treated by surgical interference to prevent renal destruction by back-pressure or by the occurrence of infection.

3. Ureterocele can, and should, be dealt with by per-urethral methods, except in the presence of complications which require open operation.

4. Diathermy applied by a cystoscopic electrode is the method of choice, and is perfectly safe providing the following rules are observed: (a) Coagulation should be effected well clear of the ureteric orifice and its papilla.

(b) An attempt should be made to perforate the cyst wall at the time of treatment. (c) Should the papilla become involved by accident and the cyst be unperforated, inflammatory occlusion becomes almost a certainty, and a small area of the wall should be excised by cystoscopic scissors, or perforated by a cystoscopic knife.

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# THE ADRENO-GENITAL SYNDROME ASSOCIATED WITH CORTICAL HYPERPLASIA; THE RESULTS OF UNILATERAL ADRENALECTOMY.

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## WITH HISTOLOGICAL NOTES

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THE association of lesions of the adrenal cortex with hirsutism and virilism in females has now become well established under the designation of the 'adreno-genital syndrome'. The pathological lesion in the adrenals may be either diffuse cortical hyperplasia or neoplastic. The latter may be either benign (adenomatous) or malignant. Both the tumour varieties are usually referred to as hypernephromata, which must be distinguished from the so-called hypernephromata of renal origin, the Grawitz tumours, which are not associated with sex changes. There are many cases on record of the adreno-genital syndrome due to tumours, but less attention has been paid to the form associated with adrenal cortical hyperplasia. Rolleston (1925),<sup>1</sup> Schwarz (1927),<sup>2</sup> Strauss (1926 and 1928),<sup>3</sup> Mathias (1929),<sup>4</sup> Grotz and Huhne (1929),<sup>5</sup> Goldzieher (1929),<sup>6</sup> and Bauer (1930)<sup>7</sup> have, however, clearly shown that this is a very definite clinical entity.

The influence of the adrenal lesion, as Mathias<sup>4</sup> has pointed out, may manifest itself in different ways, depending on two main factors—the type of lesion, and the age of the individual at its onset. Hyperplasia, owing to its slow growth, is not likely to produce such rapid changes in the sexual sphere as malignant tumours, whilst the course of the benign adenomata is intermediate between the two. The malignant tumours, moreover, are usually rapidly fatal, so that radical developmental changes outside the sexual sphere are seldom found. The age of the individual when the adrenal condition develops also plays a considerable part in determining the clinical picture. As can readily be understood, the effects of the lesion will vary according to whether the organism at the time of onset is immature or fully developed.

**Classification.**—A classification of the adreno-genital syndrome into three main types has been suggested by Schneider<sup>8</sup> and Schmidt.<sup>9</sup> This classification has been based on the above theoretical consideration.

1. *Adrenal pseudo-hermaphroditism*, the most complete form, where the adrenal changes set in during intra-uterine life. Bodily development and

external sex organs of the male type are here found associated with internal sex organs of the female.

2. *Adrenal precocity*, with or without changes in sex character, where symptoms usually develop early and within the first decade.

3. *Change of sex character after maturity*. In this form the development of the genitalia cannot be influenced to the same extent, and a group of symptoms appears which is generally included under the term 'adrenal virilism or hirsutism' (Apert<sup>10</sup> and Guthrie and Emery<sup>11</sup>). The fully developed syndrome consists of amenorrhœa and psychical changes of a masculine type, obesity, especially limited to the abdomen and trunk, or abnormal muscular development with hypertrichosis also of the male type and particularly a growth of moustache, beard, and whiskers.

According to the literature, it would appear that adrenal pseudo-hermaphroditism is usually found with diffuse bilateral cortical hyperplasia (Glynn<sup>12</sup> and Goldzieher). Noteworthy instances are the cases of Marchand,<sup>13</sup> Quinby,<sup>14</sup> and Feldmann.<sup>15</sup> Tumours, however, have occasionally been met with in this type (Spehlmann,<sup>16</sup> Seabell,<sup>17</sup> and Krabbe<sup>18</sup>). The late form, adrenal virilism or hirsutism, may also be found either with cortical hyperplasia or neoplasm. Full reference to the latter variety, which is apparently more common, will be found in the papers of Bullock and Sequeira,<sup>19</sup> Glynn,<sup>12</sup> Gordon Holmes,<sup>20</sup> and others. Bauer<sup>7</sup> (1930) has reviewed the literature of virilism with cortical hyperplasia.

There can be no doubt, therefore, of the intimate relationship between changes in the adrenal cortex and the adreno-genital syndrome. Bauer<sup>7</sup> suggests that the presence of this symptom-complex makes the diagnosis of an adrenal cortical lesion a certainty. From what has been said, it will be realized that this lesion may be either a hyperplasia of the cortex, a cortical adenoma, or a malignant adrenal hypernephroma. Any one of these pathological conditions may produce the syndrome in any of its forms. In the absence of a palpable tumour, however, and without laparotomy, it does not seem possible to diagnose the exact nature of the underlying anatomical lesion, though a consideration of the various factors mentioned above, the age of onset, and the duration and rapidity of development of the symptoms may possibly provide a clue.

**Operative Interference in the Adreno-genital Syndrome.**—Successful operative removal of cortical tumours has been reported in several instances. In the first recorded case of Thornton's<sup>21</sup> (1890), a woman of 36 with a malignant tumour recovered from operation but died two years later with an intraperitoneal recurrence. The successful removal of non-malignant tumours in adults has been recorded by Bovin<sup>22</sup> (1909), Gordon Holmes<sup>20</sup> (1925), Murray and Simpson<sup>23</sup> (1927), and Trubshaw<sup>24</sup> (1928); in children, by Collett<sup>25</sup> (1924) and Dingwall-Fordyce and Evans<sup>26</sup> (1929). In all these patients the tumour was palpable before operation. The case recorded by Gordon Holmes is particularly interesting, for here a large, non-malignant, and slowly-growing tumour of the cortex of the right adrenal was associated with changes in the sexual organs (atrophy of the uterus, disturbances in the secondary sexual characteristics), atrophy of mammae, changes in the distribution of fat and masculine appearance of limbs, and psychical changes, all of which disappeared

within a relatively short time after removal of the tumour and left the patient again an apparently normal woman. Up to the present there appear to be no records in the literature of the effects of operative interference in the adreno-genital syndrome due to adrenal cortical hyperplasia.

### UNILATERAL ADRENALECTOMY IN ADRENAL CORTICAL HYPERPLASIA.

In the present paper we propose to put on record the results of unilateral adrenalectomy in the case of three individuals representing three different clinical varieties of the adreno-genital syndrome: (1) Adrenal pseudo-hermaphroditism; (2) Adrenal virilism; (3) Achard-Thiers syndrome.

These three cases were previously submitted to laparotomy at which adrenal hyperplasia was found. In *Case 1* an illustration is provided of the most complete form of the syndrome adrenal pseudo-hermaphroditism, where changes occur before the bodily form and sex organs have become differentiated. *Case 2* represents the later variety, adrenal virilism, where the adrenal changes set in after puberty. In both instances alterations in bodily form and in the external sex organs were found, associated with hypertrichosis of the masculine type, but the function of the sex organs was disturbed to a different degree. In *Case 1* normal feminine development had not taken place and primary amenorrhœa was a prominent symptom. In *Case 2*, owing to the later development of the adrenal lesion, the normal bodily changes of puberty and a period of normal menstruation were followed by regressive changes in both spheres, bodily changes towards masculinity, a diminution in the size of the breasts, and a diminution in the menstrual flow. In both cases the adrenal lesion appears to have been of primary importance, the age of the patient at the onset of the condition accounting for the differences in the clinical syndrome produced.

*Case 3* represents a somewhat different type, in which the adrenal lesion appears to be only one element in a pluriglandular disturbance. This patient presented the symptom-complex described by Achard and Thiers<sup>27</sup> as 'diabetes of bearded women', a condition in which they found at autopsy adrenal cortical hyperplasia associated with changes in other ductless glands—namely, ovarian sclerosis and atrophy, colloid hypersecretion in the thyroid, cirrhosis of the liver, and pericanalicular sclerosis of the pancreas with increase in the islets of Langerhans, the pituitary being normal. Achard and Thiers described a case of their own and collected six others from the literature, all of which had been verified by autopsy. The chief symptoms were hypertrichosis of the face of the masculine type, without other signs of virilism, breasts normal, external genitalia normal, obesity, glycosuria with a decreased carbohydrate tolerance, hypertension, and usually amenorrhœa or irregular menstruation. These authors were of the opinion that the adrenal changes played an important part in the etiology of this condition, but were probably not primary. *Case 3* appears to be a typical example of this syndrome in a young girl.

Full details of these three cases will be found in *Tables I* and *II* and the appended case histories.

Table I.—DETAILS OF THREE CASES OF ADRENAL CORTICAL HYPERPLASIA.

TYPE	AGE	ONSET OF CONDITION	RELATION TO ONSET OF PUBERTY	DISTRIBUTION OF HYPERTRICHOSIS	FIGURE
Case 1	22	Always virile. Hypertrichosis at 14	Pre-pubertal	Masculine type	Masculine and muscular
Case 2	22	16	Post-pubertal	Masculine type	Masculine and muscular
Case 3	18	16	Post-pubertal	Feminine on body, masculine on face	Feminine and obese (trunk and abdomen)

Table II.—RESULTS OF OPERATION—TRANSSTHORACIC ADRENALECTOMY.

TYPE	B.P.*	ADRENAL REMOVED	SIZE OF ADRENAL	HISTOLOGY	MENTRICATION	INTERNAL GENITALIA	EXTERNAL GENITALIA	LATE RESULTS
Case 1	Pseudo-hermaphroditism $\frac{120}{a} \frac{78}{b}$	Left 13.8.28	$\dagger 75 \times 75$ mm.	Hypertrophy of deeper layers with much pigment present	Primary amenorrhœa	Uterus, cervix and vagina small and infantile. Ovaries apparently normal	General topography female. Clitoris much enlarged with well-formed glands. Labia diminutive	Slight change in male and female hair. Slight development of breast tissue. Otherwise no change
Case 2	Virilism $\frac{110}{a} \frac{60}{b}$ $\frac{120}{b} \frac{70}{70}$	Left 25.10.30	$\dagger 75 \times 35$ mm. Weight 12.5 gm.	Hypertrophy of deeper layers. Less pigment with finer granules	Commenced at 13 Regular 3-4 Scanty last year	Uterus, cervix and vagina infantile. Both ovaries cystic, right more so than left.	General topography female. Clitoris enlarged. Labia small	1.3.31. Falling out of hair all over body within three months of operation. Menstruation regular
Case 3	Achard-Thiers type $\frac{140}{a} \frac{90}{b}$ $\frac{140}{b} \frac{80}{80}$	Right 20.10.30	$\dagger 45 \times 30 \times 15$ mm.	Differs very little from normal	Commenced at 14 3-4 28 Ceased 20 months ago	Uterus, cervix and vagina infantile. Both ovaries cystic	General topography female. No great change	2.3.31. No change

\* a, Before operation; b, After operation.  
 † Normal size and weight varies with age. Goldzieher 1-20 years,  $46 \times 33 \times 8$  mm.; 20-40 years,  $45 \times 33 \times 9$  mm.  
 ‡ Right and left about the same, 6.5 gm. for each.  
 § Combined weight 10.6 gm.

## CASE REPORTS.

*Case 1.*—Miss L. M. Adrenal pseudo-hermaphroditism.

**HISTORY.**—Age 22. Sent for examination to Dr. Gordon Holmes, to whom we are indebted for the following notes, because at the age of 22 menstruation had not appeared. At the age of 14 she began to grow a beard and moustache, and there was excessive hairiness of the abdomen and limbs. At 16 she began to suffer from periodic headaches of the migrainous type, which have continued up to the present time. These headaches are usually accompanied by giddiness and occasionally by vomiting. Otherwise she has been free from illness.



FIG. 411.—*Case 1.* Adrenal pseudo-hermaphroditism. Muscular masculine build, absence of breasts, male distribution of hair to umbilicus.



FIG. 412.—*Case 1.* Facies with heavy beard and moustache.

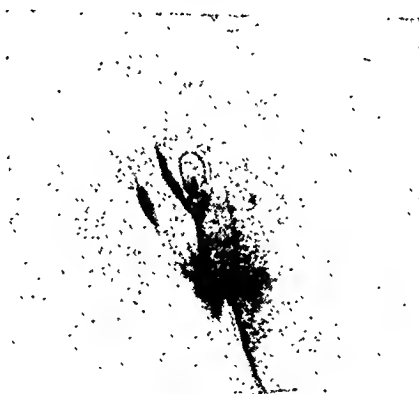


FIG. 413.—*Case 1.* Topography of external genitalia. Enlarged clitoris with glans.

**ON EXAMINATION.**—The patient is short, well developed, and muscular, her general build resembling that of a boy rather than a girl (*Fig. 411*). The breasts are undeveloped and of the masculine type, the chest broad, flat, and deep. The shoulders are broader than the hips, and the muscular development of the limbs is pronounced. The curves of the thighs and elbows stand out prominently. The most noticeable feature of this examination is the excessive hairiness: there is a heavy beard and moustache (*Fig. 412*), the pubic hair is thick and extends up the abdomen beyond the umbilicus, and the limbs are covered with dark hairs of the masculine type. Hairs are profuse in the axilla and also on the anterior aspect of the thighs and legs. Her voice has a low pitch and is somewhat masculine in



character. Her mental outlook appears to be that of a normal girl of this age, except that she admits to little interest in members of the opposite sex, and she is shy and retiring and acutely sensitive of her deformity. The general topography of the external genitalia is female, but the clitoris and glans are much hypertrophied and the labia are small (*Fig. 413*).

*Routine examination* showed no abnormalities. The cardiovascular system was normal and the blood-pressure 120/78. X-ray examination of the sella turcica revealed no evidence of any pituitary abnormality.

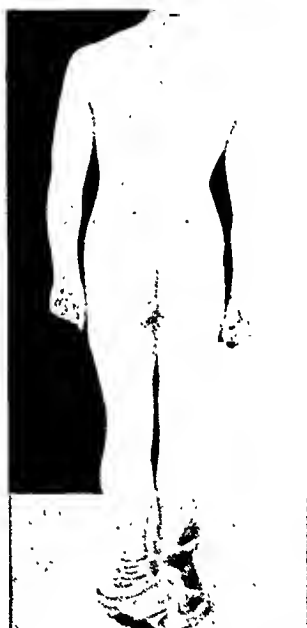
*Exploratory laparotomy* showed that the left adrenal was considerably enlarged. Notes as to the condition of the internal genitalia will be found in *Table II*.

**Case 2.—Miss S. Adrenal virilism and hirsutism.**

**HISTORY.**—Age 22. Sent for examination for excessive growth of hair on the face, body, and limbs. This began at the age of 16, three years after the onset of



**FIG. 414.**—*Case 2.* Adrenal virilism. front view. Masculine muscular build, small breasts, male distribution of hair, especially on the chest.



**FIG. 415.**—*Case 2.* The same case as *Fig. 414*, back view. Hair especially thick between the shoulders, on the small of the back, and on the limbs.

menstruation. Apart from diphtheria at the age of 17 and a mastoid operation at 20, she had been in fairly good health, though she had complained during the past few years of periodic headaches of the migrainous type. These headaches were not particularly associated with the menstrual periods. Menstruation began at 13 and had always appeared regularly at monthly intervals, though for the past twelve months the flow had become very scanty. The patient had also remarked on a diminution in the size of both breasts, starting about the same time, although previously they had been well formed and normal. Before the development of hair on her body, she had been somewhat heavier than she is now, weighing about eleven stone at the age of 16, as against nine and a half stone now. Her mental outlook, she thinks, is feminine, though she takes no particular interest in members of the male sex. She is obviously extremely shy and retiring and self-conscious about her condition.

ON EXAMINATION.—She is a short, muscular individual of definitely masculine bodily type (*Figs. 414, 415*). There is a mass of dark curly hair on the head, and the face presents a well-marked beard and moustache which she habitually shaves. Otherwise, the facial expression is feminine and the complexion pink. The voice is normal and feminine. The body and limbs are thickly covered with hairs of the masculine type: their distribution can be seen in *Figs. 414, 415*. The general topography of the external genitalia is feminine, but the clitoris is hypertrophied and the labia are small.

*Routine examination* of the various organs showed no abnormality. The cardiovascular system was normal and the blood-pressure 110/60. X-ray examination of the sella turcica revealed no abnormality. A uroselectan pyelogram showed good filling of the calices of the pelvis of both kidneys. Blood-cholesterol 203 mgrm. per cent. Sugar tolerance somewhat increased. The following blood-sugar figures were obtained at half-hourly intervals after 50 gm. of glucose: 0.070, 0.090, 0.115, 0.110, and 0.105.

*Exploratory laparotomy* revealed a marked enlargement of the left suprarenal. The right kidney appeared lobulated, especially at its upper pole. The right suprarenal was normal in size. Notes as to the condition of the internal genitalia will be found in *Table II*.

*Case 3.*—Miss E. N. Obesity and amenorrhœa with hypertrichosis (Achar-Thiers type).

*HISTORY.*—Age 18. Sent for investigation for hypertrichosis, obesity, and amenorrhœa. Enjoyed good health until two years ago. Menstruation commenced at 14 and was regular for two years. At the age of 16 menstruation ceased and the patient suddenly became excessively fat, gaining two and a half stone in a comparatively short period. No extrinsic exciting factor could be found to account either for the amenorrhœa or the obesity. During the past two years she has also complained of periodic headache of the migrainous type. She has also suffered from sudden and acute attacks of pain in the upper part of the abdomen on the right side. During these attacks she has been repeatedly examined by a doctor, but no definite evidence of an abdominal lesion could be found. During the past two years a growth of hair has appeared on her back, arms, and legs; this is especially marked on the anterior aspect of the thighs.

ON EXAMINATION.—She is a short, fat individual of feminine build (*Fig. 416*). Her obesity is entirely limited to the trunk, especially the breasts and abdomen. The breasts are well developed. There are a number of pink striae distensæ over the lateral regions of the abdomen, in the groins, and on the anterior aspects of the thighs. In all situations the skin is noticeably dry and rough, especially that of the limbs, the distal portions of which are of a peculiar purplish and mottled hue. There is a growth of side-whiskers on her face, and, extending over the back and shoulders down to the buttocks, a mantle of soft downy hair. The limbs are also excessively hairy. The distribution of the pubic hair is of the female type. The voice is feminine, and the mental outlook feminine though rather childish.

*Routine examination* showed no abnormality, though the blood-pressure was somewhat high, 140/90. X-ray examination of the sella turcica showed a normal pituitary fossa. The urine was occasionally found to contain traces of sugar. The



*FIG. 416.*—*Case 3.* Achar-Thiers type—obesity and hirsutism, side view. Note obesity limited to trunk. Downy blanket of hair over shoulders and on limbs. Mutton-chop whiskers (not seen).

sugar-tolerance test after 50 gm. of glucose was found to be decreased. The blood-sugar figures at half-hourly intervals after 50 gm. of glucose were as follows: 0.098, 0.186, 0.218, 0.224, and 0.200 mgrm. per cent. The urine passed during the test contained sugar. A similar test after 10 units of insulin and 50 gm. of glucose gave the following figures: 0.116, 0.114, 0.108, 0.110, and 0.104. The urine during this test did not contain sugar. Blood-cholesterol 0.320 per cent. Test-meal—normal curve.

*Exploratory laparotomy* revealed an enlargement of the right adrenal, an infantile uterus, and bilateral cystic ovaries.

### HISTOPATHOLOGY.

In addition to the three cases under review, we have included an account of the histology of our original *Case 1a*, clinically exactly similar in type to *Case 1*, chiefly to show a section of the lipoid granules in the outer zones of the cortex when stained with Schiarlach.



FIG. 417.—*Case 1a*. Surface of left adrenal. *a*, Central vein in hilum.



FIG. 418.—*Case 1a*. Cut section. Deep pigmentation of inner layers.



FIG. 419.—*Case 1a*. Histological appearance of cortex. Hypertrophy of inner zones and pigment granules. *a*, Medullary cells. ( $\times 50$ .)

*Case 1a* (Miss N.G.).—The adrenal removed consisted of a large firm solid body with a somewhat furrowed surface. It preserved the shape of the normal adrenal gland, but was brown in colour. It measured  $2\frac{1}{4}$  in. in length and 2 in. in breadth at its widest part (Figs. 417, 418).

On section across its centre (Fig. 418) it preserved the general structure of an adrenal gland, though the medulla could only be seen in a small area where the gland reached its greatest thickness. It was noticeable also that the colour of the cut surface was a much deeper brown than that of the outer surface, the dark area of the gland comprising the whole of the cortex except the surface layer, which was rather less than 1 mm. in thickness. Histologically (Fig. 419), the

structure did not differ from normal adrenal gland in its main features. The

most marked abnormality consisted in the amount of brownish pigment granules in the cells of the deeper layers. Microscopic examination confirmed what was evident on naked-eye examination, that four-fifths or more of the thickness of the cortex was pigmented, whereas, in the normal adrenal only the innermost third or quarter of the cortex, i.e., the zona reticularis, is pigmented. If we may distinguish this zone by the presence of pigment, it is evident that in the gland of *Case 1a* there had been an enormous hypertrophy of the zona reticularis, with little or no alteration in the size of the other parts of the cortex or of the medulla.

This pigment appeared in frozen sections stained with hæmatoxylin as very fine rounded granules of dark sepia colour which lay among the lipid droplets of the cell, or were present alone in cells which contained no lipid. It possessed a certain affinity for Scharlach. With this dye, the granules appeared larger and more yellow in colour, but they did not take on the bright brick-red colour of the ordinary lipoids of the cortex, nor were they, like the latter, doubly refractile. In paraffin sections, the pigment was still present although rather lighter in colour.

Another slight abnormality in this gland was the finding that the cortical lipid was much more predominant in the outermost, non-pigmented layer than deeper in the cortex. This can be seen in *Fig. 420*.

The medulla was of normal appearance microscopically. It formed a thin, almost continuous layer which was encroached on here and there by the cortical cells.

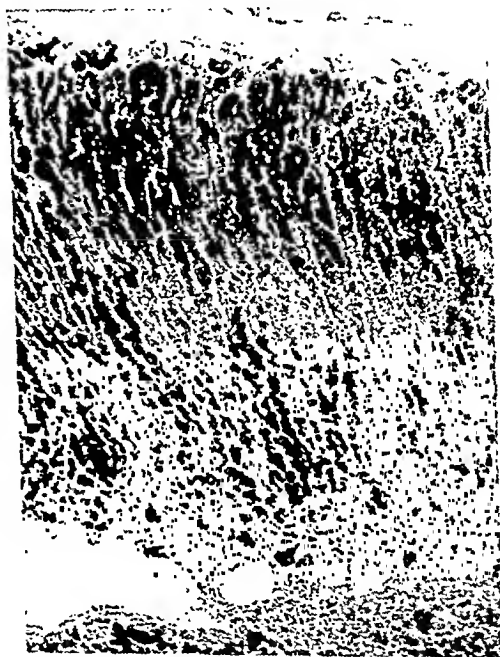


FIG. 420.—*Case 1a*. Cortex of suprarenal stained by Scharlach R. Lipoid granules chiefly in outer zone of cortex.

*Case 1.*—This adrenal had the same general appearance as that of *Case 1a*, but was even larger, measuring  $2\frac{1}{2}$  in. both in transverse and in longitudinal diameters. It consisted of two lobes united by a wide bridge at the upper part of the gland, and separating as they passed downwards. The surface was somewhat nodular (*Fig. 421*). It was of a similar dark-brown colour to that of *Case 1a*. On section, the deeper layers were all pigmented, but a thin superficial zone was of the normal chrome yellow of adrenal cortex.

Histologically (*Fig. 422*), the zona reticularis was more easily distinguished from the two outer zones, both by the arrangement of its cells and by its pigmentation, which seemed to begin more abruptly at the irregular line of junction of the two inner layers. The pigment granules were larger and more numerous, so that the pigmented cells could be easily identified under a low

power of the microscope. In this case the normal lipid pigment was more evenly distributed through the three layers of the cortex, and there were very few cells, even in the deepest layers, which did not contain at least a few red-staining lipid granules. In this case, medullary tissue was very scanty.

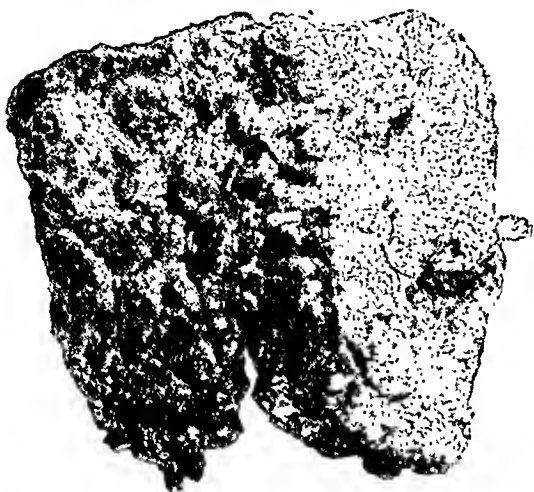


FIG. 421.—Case 1. Surface of left adrenal.



FIG. 422.—Case 1. Histological appearance of cortex. Enlargement of zona reticularis, with pigment granules. ( $\times 50$ .)

*Case 2.*—In this gland (*Fig. 423*) the zona fasciculata is better developed, being about equal in thickness to the zona reticularis (*Figs. 424, 425*). Both zones are of rather irregular width; where one is widened the other is narrowed, so that the width of the



FIG. 423.—Case 2. Surface and cross-section of left adrenal.

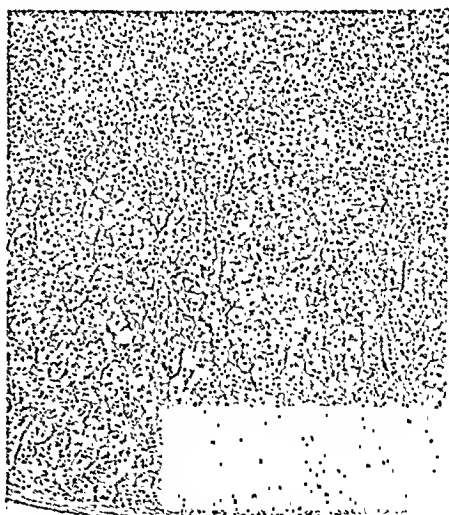


FIG. 424.—Case 2. Histological appearance. ( $\times 50$ .)

cortex as a whole is not affected. Similar brownish pigment (*Fig. 426*) is

present in most of the cells of the zona reticularis, but it occurs in finer granules and is rather less abundant than in *Case 1a*, and much less than

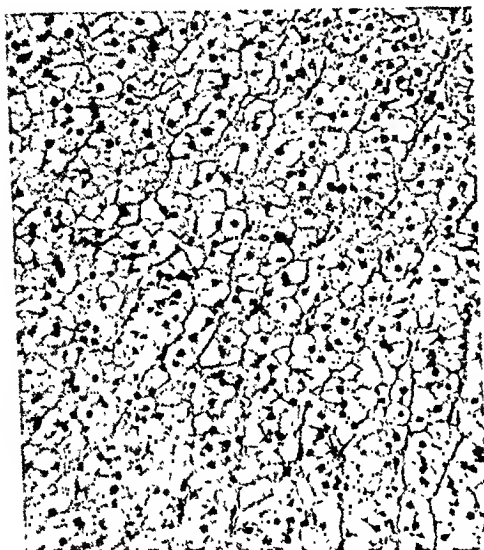


FIG. 425.—*Case 2*. Histological appearance. Higher magnification.

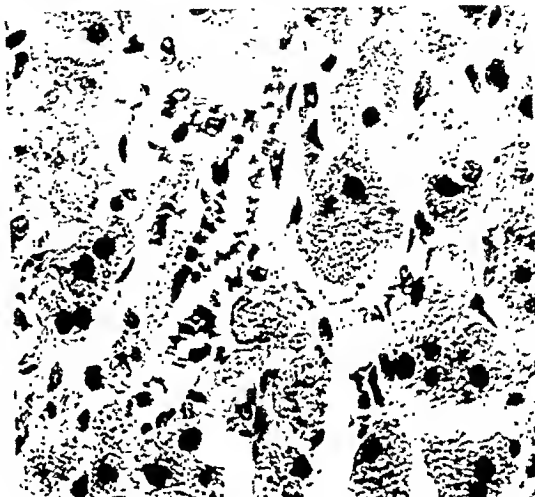


FIG. 426.—*Case 2*. Pigment granules in cells of zona reticularis of adrenal cortex.

in *Case 1*. Indeed, the only abnormality seen in sections of this gland is an abnormal width of the zone of cells which contain the pigment.

The medulla is relatively narrow, and is encroached on in many places by an ingrowth of clumps of cells from the zona reticularis.

*Case 3* (Figs. 427–429).—Histologically, this gland differs very little from

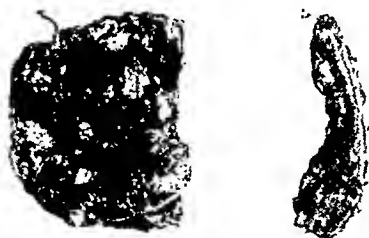


FIG. 427.—*Case 3*. Surface and cross-section of right adrenal.

the normal. The relative width of the three cortical zones is about average.

The only abnormality consists in the presence, in the cytoplasm of the cells of the zona reticularis, of small, irregular, highly refractile, dark granules of rather varied size and shape, which bear little resemblance to the pigment

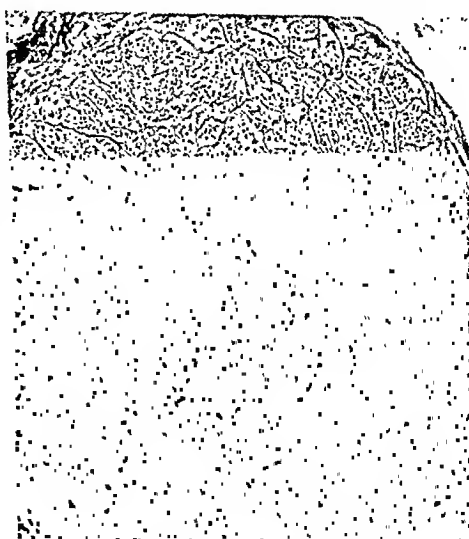


FIG. 428.—*Case 3*. Histological appearance. Islands of medullary cells in cortex (dark). ( $\times 50$ .)

granules which have been described. The difference in the method of fixation in the pigment granules. Certainly the

This appearance may result from the having produced a chemical change in the facts that these highly refractile granules are not seen either in the medulla or in the outer cortical layers, and that no pigment granules of a more usual type are seen in the cells of the zona reticularis, are in favour of this assumption.



FIG. 429.—Case 3. Histological appearance. Higher magnification.

**SUMMARY OF HISTOPATHOLOGICAL FINDINGS.**—In Cases 1, 1a, and 2 the enlargement of the adrenal appeared to be due to a simple hyperplasia of the zona reticularis of the cortex. Whether or not the medulla was also larger than normal could not be observed by the methods employed. It was, however, so scanty in the parts of the glands which were examined histologically, that it seems almost certain that it had not shared in the hypertrophy.

### OPERATIVE TREATMENT.

The symptom-complex in these cases was so characteristic that operative interference was considered justifiable, a preliminary laparotomy being performed to determine the type and site of the adrenal lesion. In the absence of a palpable tumour this preliminary operation is always advisable, as it enables the abdominal and pelvic cavities to be thoroughly explored to exclude the presence of accessory adrenal tissue or other abnormality, for in some cases hypertrichosis has been described in conjunction with tumours of the ovary. Preliminary radiological examination of the renal pelvis after the injection of uroselectan may also be undertaken in these cases, as it sometimes provides additional evidence of the presence of an adrenal tumour. This examination was carried out in two of these cases, in both instances with negative results.

The removal of a hypertrophied adrenal in an adult by the abdominal route has not been found to be a feasible operation. Being flat and embedded in fat, it is difficult to locate. Its anatomical position is by no means constant, and it has been found adherent along one surface either to the crus of the diaphragm, the wing of the diaphragm, or the upper pole of the kidney.

The second stage, transthoracic adrenalectomy, has been carried out after an interval varying from ten days to three weeks. The accompanying artificial pneumothorax may cause considerable respiratory embarrassment, and the lung usually takes from two to three weeks to re-expand. Intratracheal gas and oxygen, with a small amount of ether or ethylene, has been used, with a satisfactory absence of post-operative pulmonary complications.

A long oblique incision is made over the rib lying directly over the gland, and this rib is fractured at its neck. The incision is then carried directly through the intercostal structures below this rib. As the parietal pleura is divided, the lung collapses conveniently out of the way, the wound is retracted, and the pleural cavity packed. The diaphragm is divided in a radial direction. If this incision is kept well back, it avoids opening the peritoneal cavity. The adrenal is then removed and the diaphragm and wound closed.

**Results of Operation.**—All three cases reported in this paper made normal post-operative recoveries. Details of subsequent progress are as follows:—

*Case 1* was operated on in August, 1928, and has been carefully observed since then. Eight days after operation she menstruated for two days for the first time, and again in September, 1930, after a course of œstrin injections. There has been a slight increase in the breast tissue. Otherwise there has been no change.

*Case 2* was operated on in October, 1930. She was last seen in June, 1931. There has been a rapid shedding of hair. The chest, which was thickly covered, is now bare, and clusters of hair can be pulled out without causing any pain. The menstrual periods since the operation have been regular in time and of three days' duration.

*Case 3* was operated on in October, 1930. There has been no change since operation. The blood-pressure has remained on the same level.

The differences in the results of operation in these three cases would appear to be due to two main factors: first the age of the individual at the time of onset of the adrenal condition; and, secondly, the nature of the lesion. In *Case 1*—pseudo-hermaphroditism—the adrenal changes probably set in during foetal life before the bodily form and sex characters had been anatomically differentiated, and to some extent were no doubt bilateral. In *Case 2*—virilism—on the other hand, the adrenal changes developed later. In this instance they appear to have been initiated shortly after puberty at a time when somatic and sexual differentiation had already taken place. The adrenal lesion manifested itself by regressive changes towards masculinity and was apparently unilateral, a fact which probably accounted to a large extent for the beneficial results of operation. In *Case 3*—an example of the Achard-Thiers syndrome—evidence of adrenal change was less marked and the lesion was probably bilateral. There is evidence to suggest, however, in this condition that the adrenal cortical hyperplasia is only one element in a pluriglandular disturbance, and, moreover, probably of a secondary nature.

### CONCLUSIONS.

1. Unilateral adrenalectomy has been performed in three cases representing three different types of the adreno-genital syndrome associated with adrenal cortical hyperplasia—namely, pseudo-hermaphroditism, virilism or hirsutism, and the Achard-Thiers syndrome (diabetes of fat bearded women). All three patients have made complete recoveries and are in good health at the present time, three years, ten months, and ten months respectively after operation. In no instance were untoward post-operative symptoms observed, and the blood-pressure did not vary during or after operation.



2. The effects of unilateral adrenalectomy on the main symptoms of the syndrome, the hypertrichosis, and sex changes, have not been the same in the three cases. In the late syndrome—adrenal virilism—markedly beneficial results were obtained, but in the early form—pseudo-hermaphroditism—and in the Achard-Thiers type—probably a pluriglandular disturbance—no change in the symptoms was observed. Our experience would, therefore, suggest that unilateral adrenalectomy is indicated in the late syndrome—virilism—due to adrenal cortical hyperplasia, where the adrenal lesion is mainly unilateral, but not in the other two forms.

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## EMBRYONIC EPITHELIAL RESTS IN THE PITUITARY.

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THE origin of many hypophysial tumours has been traced to embryonic rests, and yet these foetal remnants have been but rarely investigated. Luschka (1860) was the first to describe groups of squamous epithelial cells in the pituitary. In 1903 Erdheim, in the course of an investigation of the endocrine glands, noted a number of epithelial rests in an hypophysis included in the series he was investigating. This led to a further investigation in the following year, when he examined 13 adult pituitaries in serial sections. Of the 13 glands, he found that 10 contained embryonic rests, and their distribution was most commonly along the anterior surface of the infundibulum, and beneath the capsule of the upper surface of the anterior lobe. The connection these rests bear to hypophysial tumours has been demonstrated on a number of occasions. Jackson (1916) collected 38 cases, to which Duffy (1920) added 17, and since then a number of further cases have been described. Thus embryonic rests in the pituitary have a definite practical significance, for they may give rise to tumours or cysts.

In this series, 230 hypophyses were examined. These glands had been removed at routine post-mortem examinations whenever the brain was examined if the tissue was in a sufficiently good state of preservation. None of these 230 cases showed recognizable pituitary symptoms before death. The glands were cut antero-posteriorly through the infundibulum and fixed in Helly's fluid. Sections were prepared from each half of the gland, and stained with hæmalum and eosin. Only a few of the 230 glands were examined in serial sections.

Of the 230 hypophyses examined, 71 contained epithelial rests of an embryonic character (30·4 per cent). Of the 71 examples, squamous rests alone were found in 32 glands, primitive epithelial rests alone in 29, and primitive glandular epithelial rests in 3. In only 7 of the 71 glands containing embryonic rests were there more than one of the three possible types of rests present. However, when several types did occur in the one gland, a single rest might contain cells of all three types, and on occasion such mixed types of rests were found to be continuous with strands or groups of glandular cells of the normal adult type found in the pars glandularis.

The classification of the rests into squamous, primitive epithelial, and primitive glandular rests was based on those obvious histological features distinctive of each group in sections stained by hæmalum and eosin. Those classed as squamous had cells possessing intercellular bridges and the characteristic general appearances of squamous epithelium. The primitive rests were differentiated by the very low type of epithelium they contained. The types of cells varied from gland to gland, but were fairly uniform in the rests

of any particular gland. The primitive glandular rests were made up of closely packed medium-sized cells of a spheroidal shape and of the type usually seen in a spheroidal-cell carcinoma of the glandular type (*Fig. 436*). The appearances definitely suggested that the tissue was glandular, although primitive. These features may have resulted from anaplasia.

*Table I.*—DISTRIBUTION OF EMBRYONIC REST IN THE 71 CASES.

PART INVOLVED	NO. OF GLANDS	NO. WITH SQUAMOUS RESTS	NO. WITH PRIMITIVE EPITHELIAL RESTS	NO. WITH PRIMITIVE GLAND RESTS
Capsule of pars anterior ..	30	11	17	3
Pars anterior .. ..	6	5	2	0
Capsule of pars intermedia ..	5	3	2	0
Pars intermedia .. ..	12	9	3	0
Capsule of infundibulum ..	5	3	2	0
Infundibulum .. ..	19	15	4	0
Capsule of pars posterior ..	9	3	6	0
Pars posterior .. ..	2	0	0	2

*Table I* shows that the most common site for these rests was the capsule of the anterior division of the pituitary (*Fig. 435*), while the infundibulum and the pars intermedia (*Fig. 438*) had a somewhat lower incidence. Two or more parts of the gland were involved in 15 cases. Rests were present in the infundibulum and pars intermedia of 4 cases, and in the infundibulum and the capsule of pars anterior in 3. These combinations can be expected by reason of the developmental process through which the gland passes in embryonic life. In the remaining 8 glands the combinations of sites showed an accidental grouping which lacked any significance.

**Age Distribution.**—An investigation of this character is hardly complete without an analysis of the age distribution amongst the cases of this series. In *Table II* the age distribution in the 71 cases is compared with that in the 218 of the 230 pituitary cases in which the age was available.

*Table II.*—AGE DISTRIBUTION IN THE 71 CASES.

AGE-GROUP	INCIDENCE IN 218 CASES OF THE PITUITARY SERIES	INCIDENCE IN SQUAMOUS REST CASES	INCIDENCE IN PRIMITIVE EPITHELIAL REST CASES
	Per cent	Per cent	Per cent
1-9	7	0	0
10-19	10	3	3
20-29	13	8	16
30-39	13	20	13
40-49	22	36	30
50-59	20	20	23
60-69	10	6	10
70 and over	4	6	6
Totals	218	35	31

This analysis tends to show that the incidence is highest in the age-group 40-49, but the same is true in the general series of 218 pituitary cases.

Although of no great significance, it ought to be noted that few rests were found in subjects under 20 years of age, although the general pituitary series included 17 per cent in this category. If the rests are of embryonic origin and not groups of cells which have undergone metaplasia, then one may reasonably expect the rests to occur at all ages with an incidence parallel with that seen in a general series of pituitary cases. On the whole, however, the age distribution in this series discloses little of any importance. ]

## DISCUSSION.

To grasp the full significance of the above findings, a brief outline of the embryology of the pituitary is essential. Rathke's pouch, from which the pituitary originates, begins as a small pit in the stomodæum of the early embryo. The pouch penetrates more deeply, and later becomes closed off to form the hypophysial cavity. At the same time the evagination of the infundibulum takes place from above. Thus, the hypophysis at this stage is an elongated sac with one side in contact with the infundibulum which gives rise to the pars intermedia, and the other side produces the pars anterior. This embryonic tissue, which is to become the pars anterior, is situated on the anterior aspect of the hypophysial cleft at its lower extremity, and development occurs in an anterior and superior direction, so that eventually the anterior hypophysial cell mass comes into contact with the hypophysial stalk. The infundibulum is a downward extension of the third ventricle, and that area of the infundibulum in contact with the pituitary becomes the pars nervosa (*Fig. 430*).

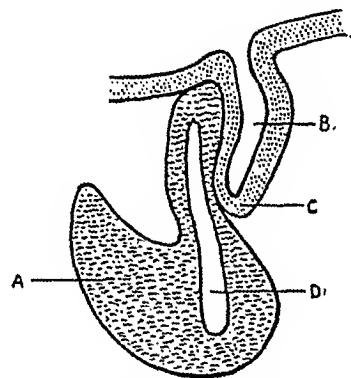


FIG. 430.—To illustrate the chief embryological features in hypophysial development. A, To form pars anterior; B, Cavity of infundibulum; C, To form pars nervosa; D, Hypophysial cavity.

According to the embryonic development of the gland we may therefore expect to find embryonic rests frequently: (1) In the capsule of the pars anterior, because the primitive shell of the pituitary would occupy, relatively speaking, a capsular position as the epithelial portion appears to be distributed peripherally at this early stage; and (2) In the infundibulum as well as in the pars intermedia where a great part of the primitive gland develops. The findings in this investigation, as already shown, are in accordance with these observations. Both the primitive epithelial rests and the squamous rests had similar distribution, with the exception that the primitive rests were less numerous in the infundibulum.

Amongst the embryonic rests there were a few features worthy of some consideration. In a number of glands, small rests were scattered quite evenly along the anterior wall of the infundibulum, a feature which is easily understood but rather picturesque (*Fig. 431*). Of greater interest were the crescentic rests which more or less covered the upper surface of the anterior pituitary. They were composed of very primitive epithelium and were continuous with the capsule. The type only occurred in three glands (*Fig. 432*).

Some investigators have reported adamantinomata in the hypophysial region, but in the present series no rests of a corresponding type were noted. An appreciable number of the primitive rests consisted of spindle cells and



FIG. 431.—Squamous rests scattered along the anterior surface of the infundibulum. ( $\times 70$ .)



FIG. 432.—Portion of a crescentic rest covering the whole of the upper surface of the anterior pituitary. It is made up of small primitive epithelial cells, and the rest is continuous with the hypophysial capsule. ( $\times 55$ .)



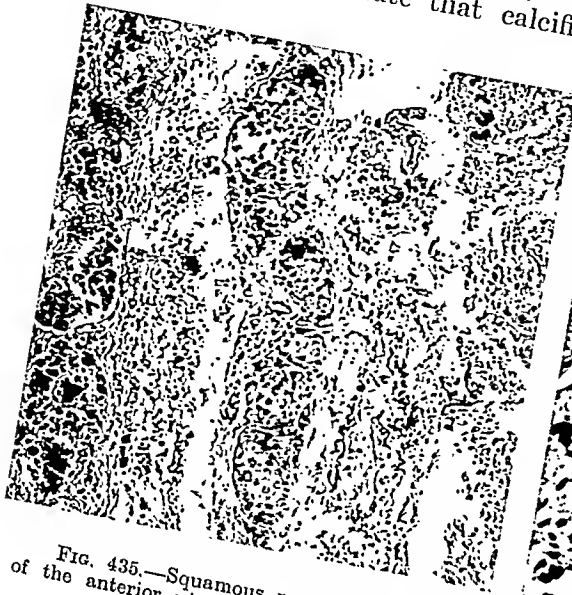
FIG. 433.—Squamous rest in the anterior pituitary. One small group of cells has a central mass of colloid. ( $\times 85$ .)



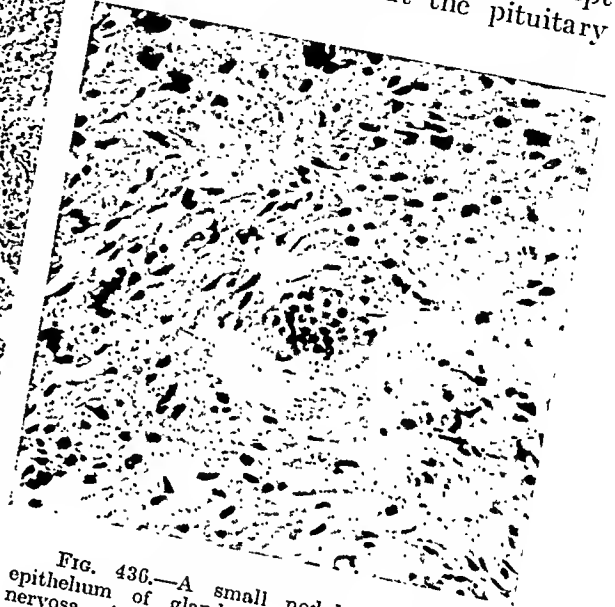
FIG. 434.—Squamous rest in the anterior pituitary. ( $\times 85$ .)

other types associated with adamantinomata, but the rests were obviously of a simple character and not adamantine. A number of rests, too, contained hyaline or colloid masses (*Fig. 433*), but none of these were to any degree

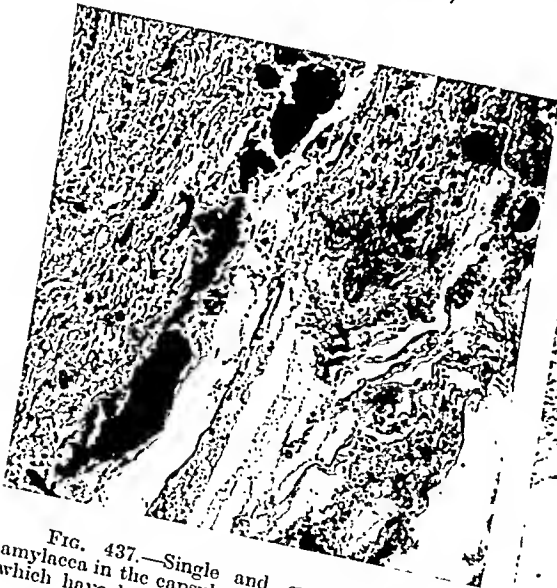
calcified, although a number of corpora amylacea about the pituitary had undergone calcification (*Fig. 437*). These are of doubtful significance, except that they may indicate that calcified material in and about the pituitary



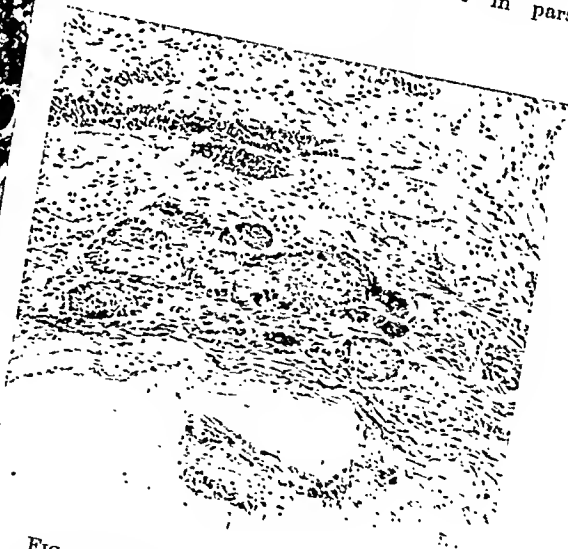
*FIG. 435.*—Squamous rest in the capsule of the anterior pituitary. ( $\times 85$ .)



*FIG. 436.*—A small nodule of primitive epithelium of glandular appearance in pars nervosa. ( $\times 170$ .)



*FIG. 437.*—Single and groups of corpora amylacea in the capsule of the posterior pituitary which have become calcified. ( $\times 85$ .)



*FIG. 438.*—Squamous rest at the foot of the infundibulum in pars intermedia. ( $\times 85$ .)

even in the presence of embryonic rests or tumours need not necessarily imply an adamantinæ source. Before concluding, there is a further and important point which must be considered. If epithelial hypophysial and suprasellar tumours originate from

these embryonic rests, as apparently they do, then these tumours may occur not only on the surface and in the stalk, but also within the substance of the gland (*Fig. 434*). Hence a tumour of embryonic origin may be intrasellar. This is of great importance, for a distended sella in a child or young person may be due to a congenital epithelial tumour even though no suprasellar calcification is evident on X-ray examination.

I wish to thank Mr. Jefferson for his very helpful interest in this work. I also wish to thank Mr. H. C. Taylor for his care in making the photomicrographs.

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## ISOLATED FRACTURE OF THE CARPAL SEMILUNAR AND KIENBÖCK'S DISEASE.

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FOLLOWING fracture, or occasionally without any definite history of injury, a progressive degenerative change which was first described by Kienböck may occur in the carpal semilunar bone. A considerable number of examples of fracture of the semilunar had already been recorded before the appearance of Kienböck's paper in 1910, most of which are summarized in Finsterer's work on isolated fracture of the semilunar bone published in the preceding year. Before the extended use of X rays, the clinical diagnosis of this lesion presented great difficulty, and it was only recognized in exceptional cases where the semilunar was fractured in association with other injuries (Peste, Hunt), or during routine dissections of the carpus by morbid anatomists such as Pfizner.

Before the appearance of Kienböck's work, all the semilunar lesions which had been described were attributed to trauma, and the term 'Kienböck's disease' was used by later writers to mark his recognition of the fact that degenerative changes occur not infrequently in the semilunar—somewhat similar to those previously described by Preiser in the scaphoid—where no definite history of adequate trauma could be elicited, and with no antecedent fracture demonstrable in the radiograms. So far as the carpus is concerned, these progressive changes *terminating in deformation* are most common and most characteristically seen in the semilunar. It is convenient and justifiable to continue the use of the term 'Kienböck's disease' for all varieties of the lesion in that bone, with the exception of primary fissured and compression fractures immediately following injury, which are better described as such, in view of their definite traumatic causation. We must bear in mind, however, that 'Kienböck's disease' is merely a convenient clinical designation for a condition which is not a distinct pathological entity.

Kienböck's work was based on sixteen personal cases and some twenty others collected from the literature. Both he and Preiser considered the bone changes to be comparable with those occurring in Kümmel's disease of the bodies of the vertebræ (spondylitis traumatica) and secondary to interference with the blood-supply, which, in the case of the semilunar, is chiefly carried in the dorsal ligaments, where it is subject to injury by temporary subluxation



or other local trauma. This hypothesis has been criticized, mainly on the ground that definite dislocation of the semilunar has not been followed by these bony changes in cases where operative reduction has been effected.

Amongst the theories advanced by later writers, that of Leriche and Buehman, by whom Kienböck's disease is considered to be a post-traumatic osteoporosis, appears to be untenable. Osteoporosis does undoubtedly occur in the carpal bones, but it differs from Kienböck's disease, in which definite areas of necrosis are shown by diffuse or patchy sclerosis in the radiograms and on histological examination. In many respects the semilunar lesion resembles the 'pressure' group of epiphyseal disorders such as Legg-Perthes' disease of the epiphysis of the head of the femur, and Freiberg's disease of the head of the second metatarsal.

Axhausen has emphasized the fact that these diseases may occur without any history of injury and regards them as infarctions due to 'benign myelotic emboli'. This view is not wholly excluded by the negative findings on bacteriological examination of the excised bone, as the local reaction might persist after the attenuated infection which caused it had died out.

Our knowledge of Kienböck's disease is mainly derived from foreign sources, as little has been written on the subject in this country. Laming Evans and Fairbank each showed a case to the Orthopædic Section of the Royal Society of Medicine in May, 1929, and later in that year Riches published his article, which was based on one case, and included a brief survey of the literature.

### CASE REPORTS.

*Case 1.*—E. R., age 21, bricklayer. (Mr. Lytle.)

Attended the Royal Infirmary in August, 1928, complaining of pain and stiffness in the left wrist, due, he said, to an injury sustained two weeks previously, when "the wrist was trapped by a plank which fell across the back of it".

**X RAY.**—The radiograms showed marked deformation of the semilunar, which was flattened and spread out in the antero-posterior axis.

**OPERATION.**—As the man was unable to work, and conservative treatment seemed hopeless in view of the advanced deformation of the bone, it was excised through a dorsal incision in September, 1928.

A claim for compensation was repudiated as no notice of accident had been given, and the radiograms suggested that the lesion was of old standing; and although the man denied any previous injury, he was known to have 'sprained' that wrist two years previously by a fall from a bicycle.

**AFTER-HISTORY.**—He resumed light work eight weeks after operation, and full work as a bricklayer in four months. He has left the district, but reported in June, 1931, that he has been able to work full time ever since, though he has some stiffness, and very occasionally some pain, after a hard day's work.

*Case 2.*—J. A. H., age 31, bricklayer. (Mr. Lytle.)

On Aug. 26, 1927, he was struck by a falling brick and fell heavily on his outstretched hands. The left wrist swelled up and became painful and was X-rayed at the infirmary three days after the accident. The radiogram was returned as showing 'no bony injury'.

**X RAY.**—The patient was treated for some months, without improvement, and radiograms repeated in December, 1927, showed absorption of the left semilunar bone about a fracture on the concave distal articular surface (*Fig. 439*). Re-examination of the first radiograms showed the presence of a fine fissured fracture in the same situation, which had been missed by the radiologist.

Prolonged conservative treatment was continued, but the symptoms persisted, and showed deformation of the bone (*Fig. 440*).

The left semilunar was excised in December, 1928, through a dorsal incision. After operation the patient was "slightly improved, as the wrist remained partly stiff, but less painful."

**AFTER-HISTORY.**—In July, 1929, he received a lump-sum settlement of £300, and returned shortly afterwards to work as a foreman bricklayer.



**FIG. 439.**—*Case 2.* Absorption of semilunar around a fissured fracture (Dec. 20, 1927).



**FIG. 440.**—*Case 2.* Deformation of semilunar (Oct. 10, 1928).

Re-examined in June, 1931: There is some restriction of the movements at the left wrist, but otherwise it appears to be normal. Finsterer's\* signs are negative. No wasting of the forearm muscles. He says that he cannot support the weight of his body on his left hand, and sometimes gets pain in the wrist. He is working fairly regularly as a foreman bricklayer, and has no difficulty in doing "work where skill is required rather than labour". He was satisfied with the result of operation, but thought that it had been delayed too long, and said that "if he broke the other wrist, he would have the operation right away".

**Case 3.**—M. G., female, age 18.

Attended the Royal Infirmary in January, 1931, complaining of pain, swelling, and stiffness of the right wrist, of about two years' duration. The trouble had commenced gradually, shortly after an attack of scarlet fever. There was no history

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\* Finsterer's signs of fracture of the semilunar: (1) Tenderness in the region of the semilunar elicited by tapping on the knuckle of the third metacarpal bone of the closed fist; and (2) Retraction of that knuckle—from vertical shortening of the semilunar—in compression fracture.

of injury. She was then a general servant, but the wrist got so painful after scrubbing floors, that she became a shop-assistant. The wrist then improved for a time, but later the symptoms returned and compelled her to give up work altogether as she was unable to handle even light weights.

She was treated with massage and hot whirlpool baths for three months from October, 1930, without improvement. In January, 1931, there was a slight amount of swelling over the back of the right wrist, with tenderness on pressure over the semilunar bone. Movements were restricted to about one half of the normal range. Finsterer's signs were negative. Wassermann negative.

**X RAY.**—The radiograms showed typical degenerative changes with deformation.

Conservative treatment was again tried for three months. Coarse intra-articular creaking on dorsiflexion was noticed in March.

**OPERATION.**—The bone was excised through a dorsal incision on April 9. It broke up and was removed in bits.

**PATHOLOGIST'S REPORT.**—No organisms found on bacteriological examination of the freshly excised bone, and cultures remained sterile. "The bone shows marked changes. The marrow is replaced by fibrous granulation tissue, in places dense and in others younger and looser. The productive osteitis and osteochondritis mentioned by Ewing (*see* Voshell) is shown (Figs. 441, 442). The condition does not now seem to be active. It appears to be a form of osteitis fibrosa probably traumatic in origin. There is no microscopic evidence of infection."



FIG. 441.—Case 3. Showing dense fibrous tissue replacing bone-marrow (A), and earlier granulation tissue (A1). At B is evident the thickening of fibrocartilage. ( $\times 5$ .)



FIG. 442.—Case 3. From the area marked A in Fig. 441. Portion of bone showing whorls of fibrous tissue and granulation tissue occupying bone-marrow. ( $\times 40$ .)

**AFTER-HISTORY.**—Intra-articular crepitus and pain persisted after operation. On Aug. 4, a newly formed fibro-osseous nucleus and a fragment of the anterior horn, which was adherent to the anterior ligament in front of the os magnum and had been missed at the first operation, were excised. Progress after the second operation was satisfactory. The patient was able to do housework in September, and resumed her ordinary work in October.

**Case 4.**—W. S., age 34, collier.

Ceased work in January, 1931, and claimed compensation for 'sprained wrist'. When seen a fortnight later he gave the following history: The left wrist had troubled him off and on for three months, but there had been no injury to it "except for knocks and bumps in the course of his work", and finally, "it went altogether, and he couldn't get the hand back to use the pick".

There was some swelling over the back of the left wrist, with maximum tenderness on pressure over the semilunar bone. Flexion and extension movements were



FIGS. 443, 444.—Case 4. Antero-posterior view of right and left wrists.



FIGS. 445, 446.—Case 4. Lateral view of right and left wrists.

limited to about one-third of the normal range. Finsterer's signs were negative. Wassermann negative.

**X RAY.**—The radiograms showed the typical deformation and sclerosis of Kienböck's disease (Figs. 443-446).

Conservative treatment was tried for three months with little improvement, and an attempt to resume work failed. Liability was repudiated as there had been no accident, and the X-ray appearances suggested that the disease was of old standing.

**OPERATION** (April 23, 1931).—The semilunar was excised through a dorsal incision. The bone fragmented during removal. Bacteriological examination of the fresh specimen was negative, and cultures remained sterile.

**PATHOLOGIST'S REPORT.**—"There are small areas similar to those in *Case 3*, with a replacement granulation tissue. The condition is, however, much more recent, and in areas where the process is active there are giant cells (osteoclasts) lying in bays and lacunae of dead bone fragments, which probably represent remains of fractured cancellous bone (Figs. 447, 448). The granulation tissue is to be found not only within the bone but also around the vessels in the cartilage, and the outer aspect of the cartilage is more firmly attached to a greater amount of denser fibrous tissue."



FIG. 447.—*Case 4*. A, Granulation tissue replacing marrow; B, Dense fibrous tissue surrounding an area where there is absorption of dead bone; C, Vascular spaces in cartilage, showing granulation tissue. ( $\times 5$ .)



FIG. 448.—*Case 4*. From the area marked B in Fig. 447. Area showing dead bony fragments undergoing absorption by giant cells. Dense fibrous tissue at the periphery. ( $\times 40$ .)

After his claim for compensation had been dismissed the patient came back to say that he remembered an old injury to the wrist. As a lad (aged 14) he worked on a farm, and whilst engaged in milking a cow, it kicked out and spun him round so that he fell heavily on his outstretched left hand. He was off work for ten days, and the wrist was treated for a sprain. He then resumed work, and two or three years later went in the pit, where he had been at the coal face for many years. The wrist had not troubled him again until three months ago. The quiescent period in this case, therefore, appears to be of nearly twenty years' duration.

**AFTER-HISTORY.**—He returned to light work on June 11, and soon after to his ordinary work. He wrote on July 29 as follows: "My wrist is going on excellently, and I can do a full day's work at the coal face, which is a severe test, as the coal is very hard. I have no pain whatever, and have a lot more action now than before my operation."

**Case 5.**—F. L. B., male, age 25, tube forger.

On Feb. 20, 1929, he was drawing a tube out of the bush, and before he had a proper grip on the tongs, the crane took off and jerked his right wrist. He finished the lift and continued to work, but went to his doctor "happen a fortnight later" because of pain in the wrist. He visited the doctor seven or eight times between

March and August, when he had to stop work, because the wrist was so painful and swollen "that he couldn't wash or dry himself".

**X RAY.**—An X ray taken in September, 1929, showed typical changes of Kienböck's disease.

The man then complained of pain in the front and back of the wrist. There was no swelling, but considerable limitation of flexion and extension. Finsterer's signs were not present.

A claim for compensation was repudiated because there was considerable doubt about the circumstances of the alleged accident.

**AFTER-HISTORY.**—He returned to light work, and continued to do that for a year, but then changed his occupation. His present condition cannot be ascertained.

*Case 6.*—J. H., age 24, trammer (pushing tubs) in pit.

On March 15, 1930, he injured his right wrist through falling forward and striking the palm against the edge of the tub he was pushing. He finished the shift and was then off work for a fortnight, while the wrist, which was painful and swollen, was treated for a sprain. He returned to work with the wrist bandaged, but had to give up in December, 1930, as the wrist was too painful to use. He had treatment for five months, then went back to work, wearing a wrist-strap.



FIGS. 449, 450.—Case 6. Antero-posterior and lateral views of right wrist.

When examined on July 13, 1931, there was localized tenderness on pressure over the right semilunar. Finsterer's signs were negative. Movements were only slightly restricted, but flexion was painful. No wasting of the forearm muscles was present.

**X RAY.**—The right semilunar is flattened and fragmented. In the lateral view it can be seen sitting over the head of the os magnum like a cap. The appearances are strongly suggestive of Kienböck's disease (Figs. 449, 450).

No claim for compensation was made, as the man found that he could still do his own work with the wrist bandaged.

*Case 7.*—W. S., age 24, collier.

About six months ago he jarred the left wrist when knocking out a pit-prop with a hammer. He had some pain for a few days, but regarded the occurrence as a trivial matter, and failed to report it. He wore a wrist-strap for two or three weeks, and continued to work on the alternate days the pit was working. He then discarded the strap and worked for five months comparatively free from pain. The

pain, stiffness, and swelling of the wrist then returned, and after he had been off work for three weeks he sought advice at a district hospital, where the wrist was X-rayed.

**X RAY.**—The semilunar is not much flattened, but it is densely sclerosed and fissured. The appearances are suggestive of Kienböck's disease.

No claim for compensation was made because of the absence of notice.

*Case 8.*—G. S., age 55, stallman in pit. (Mr. Sutcliffe, Chesterfield.)

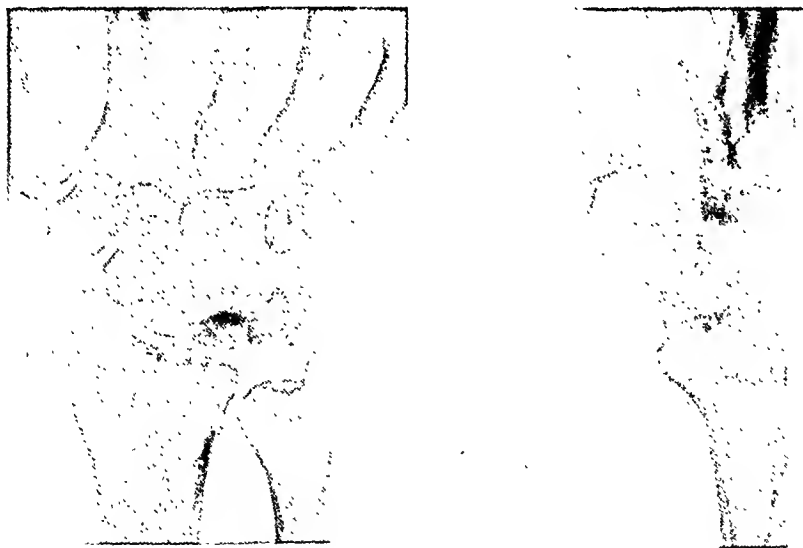
In January, 1923, he was struck on the back of the right wrist by a fall of timber. The wrist was X-rayed, but he does not think any fracture was discovered, as no splint was applied. The painful and swollen wrist was treated by rest and lead and opium applications, and later by massage. He was off work for five months. He has had some pain and stiffness in the wrist, and loss of strength in the grasp of that hand, practically ever since, and these troubles gradually got worse and compelled him to give up work two months ago.

When he first attended Chesterfield Hospital, in August, 1931, the range of movement at the right wrist was found to be diminished in both flexion and extension. The grasp was weakened, and he complained of pain in the wrist on movement and on pressure over the front and back of the carpus.

**X RAY** (Aug. 19, 1931).—"The semilunar is flattened and mottled and shows considerable sclerosis. The appearances are suggestive of Kienböck's disease with secondary osteo-arthritic changes in the styloid process of the radius, the scaphoid, and cuneiform."

*Case 9.*—S. D., age 21, collier. (Dr. Hurst, Chesterfield.)

Worked as a trammer in the pit from the age of 14, but had to stop three years ago because of pain and stiffness in the left wrist. He was off work for



FIGS. 451, 452.—*Case 9.* Bilateral Kienböck's disease: right wrist.

six months. The wrist was X-rayed and treated at Chesterfield Hospital. The patient then returned to haulage work for two years, but had pain in the left wrist, and six months ago he began to have trouble with the right wrist as well. He tried to start work at the coal face four months ago, but had to give up, as both wrists were very stiff and painful. Since then he has been on the panel, and has again

received hospital treatment without much benefit. He can remember no definite injury to the wrists or accidents of any kind.

The active and passive range of movement at the right wrist is very restricted and painful, and there is local tenderness and some swelling over the back of the carpus. The movements of the left wrist are slightly restricted, and there is local tenderness on pressure over the semilunar bone, but no swelling of the tissues.



FIGS. 453, 454.—Case 9. Bilateral Kienböck's disease: left wrist.

**X RAY.**—The radiograms showed typical changes of Kienböck's disease in both semilunars (Figs. 451-454).

**OPERATION** (Dec. 7, 1931).—Excision of the right semilunar through a dorsal incision.

**Case 10.**—G. K., age 60, collier. (Dr. Evans, Grassmoor, Chesterfield.)

In 1920 he sustained a severe blow on the back of the right wrist from a 'wringer' in the pit. He was off work for two months, and then returned to his ordinary work. Ever since then he has been troubled at intervals with pain, swelling, and stiffness in the wrist, which gradually got worse and made him give up work altogether in October last. In the earlier years he improved with rest and treatment, but is now unable to do any work which necessitates use of the injured wrist.

**X RAY.**—The radiograms show very advanced degenerative changes in the right semilunar, with osteo-arthritic changes in the neighbouring articular surfaces.

**AFTER-HISTORY.**—The patient is now receiving compensation, and has been on compensation at intervals since his accident.

**Case 11.**—S. A., age 21, housewife. (Dr. Kemp, Thurcroft, near Rotherham.)

Two years ago she slipped while washing the floor, and fell heavily on the palm of the left hand. The wrist was painful and swelled slightly, but after a few days she felt nothing of it till she began to get about after her first confinement in June, 1931. The pain then became very severe if the wrist were touched or knocked, and she could not wring out clothes or scrub floors. The wrist "seemed to lock when it was twisted, and she had to move it about to work it loose again".

When seen in October there was marked swelling over the back of the left wrist, with limitation and pain on movement in all directions.



X RAY.—The radiograms (*Figs. 455-457*) showed marked sclerosis of the left semilunar, with fragmentation and degenerative changes.



*FIGS. 455, 456.*—*Case 11.* Antero-posterior and lateral views of left wrist.



*FIG. 457.*—*Case 11.* Oblique view.

OPERATION (Nov. 5).—Excision of the semilunar through a dorsal incision. The bone fragmented and was removed in bits. The swelling of the synovial membrane around the bone was particularly marked, and the surrounding tissues were congested and œdematous.

PATHOLOGIST'S REPORT.—Semilunar bone. Cartilage necrotic and bone probably necrotic. Lacunæ contain granular unrecognizable material, apparently originally cellular—? marrow altered by decalcifying fluid—but the other bones examined did not contain marrow. At one end cortical bone appears to be in process of erosion by cellular granulation tissue growing in from without.

Case 12.—E. W., age 23, female, domestic servant. (Mr. Sutcliffe, Chesterfield Hospital.)

At the age of 12 she fell at play and fractured the lower end of the right radius. The forearm was in splints for a month. In September, 1930, she attended out-patients, complaining of pain in the right wrist which interfered with her work, and had gradually become worse during the preceding twelve months. She was treated for two months by radiant heat and rest on a splint, without much improvement.

X RAY.—The radiograms showed fragmentation and sclerosis of the right semilunar (Kienböck's disease). No sign of old fracture of radius.

OPERATION (Nov. 4).—Excision through dorsal incision.

AFTER-HISTORY (Dec. 31).—The patient reports that the pain has gone but the wrist remains stiff. She can do housework, but cannot resume domestic service as the wrist is easily tired.

### DISCUSSION.

Our twelve cases confirm in general the accepted facts as to the incidence and clinical features of the lesion. The number in males predominates by 9 to 3. The right side is more often affected, and as a rule it occurs in those engaged in the heavier forms of manual labour, between the ages of 18 and 40.

The former facts require no comment, for they are to be expected in a lesion which is usually caused by trauma, but the age appears to have a direct bearing on the occurrence of the condition, as the usual injury—a fall or blow causing forcible dorsiflexion of the wrist—is more likely to cause, in younger individuals, a separation of the inferior radial epiphysis, whilst in older people a Colles's fracture is the more frequent result of that type of accident.

The lesion is, in the great majority of cases, undoubtedly caused by injury, which may be of a comparatively trivial nature, with the result that the temporary pain and swelling of the wrist which follows is treated as a simple sprain, while even if an X-ray examination is made, the tiny fissured fracture in the bone may escape detection even by an experienced radiologist (e.g., Case 2). Massage and movements are instituted as soon as the more acute symptoms subside, without the sufficient immobilization which is so essential in carpal injuries to ensure repair of the bone lesion. The patient either continues to do his ordinary work, or returns to it too soon. The degenerative changes progress during the latent period, and the characteristic deformation is brought about gradually, from continued occupational strain, or follows some further trivial injury.

The few available serial radiograms (Case 2), and the histories in the great majority of cases, offer almost convincing evidence that the deformation is secondary and due to a progressive pathological process, initiated by trauma; and we doubt if a primary compression fracture of the semilunar bone ever does occur as an isolated lesion.

**Etiology of Kienböck's Disease.**—The semilunar bone is, from its anatomical position, liable to mechanical injury when the hand is violently dorsiflexed; and in a dissected wrist, when the dorsal ligaments are removed to expose the back of the semilunar—without interference with any of the other ligamentous structures of the carpus—in dorsiflexion of the hand, the semilunar is seen to be 'crowded out' by its neighbours which are firmly attached to the strong lateral ligaments, so that it tends to spring forward. and after removal of the dorsal ligaments\* it can be dislocated by comparatively little force on to the palmar surface. Whilst the dorsal ligaments remain intact, it keeps in position, and is subjected to stress between the head of the os magnum and the unequal counter-pressure exercised on its proximal aspect by the triangular fibrocartilage and the more resistant median articular facet and edge of the inferior surface of the radius.

In one of Müller's cases the semilunar developed an extreme degree of deformation as a probable result of the abnormal pressure strain to which it was exposed because the ulna was shortened by old disease, and that author expressed the view that even a comparatively slight degree of shortening of the ulna might be a definite etiological factor in the disease. Blencke found a slight variation of this nature in no fewer than 11 of his 43 cases.

The fact, however, that fractures of the scaphoid are comparatively common, though degenerative changes in that bone are rare, whilst the opposite is true as regards the semilunar, calls for explanation. There appears to be definite justification for the suggestion that the degenerative changes in the bone are closely associated with its *poor blood-supply*, for the bone occupies a central position in the joint and lacks the broad vascular lateral attachments of the scaphoid and cuneiform. Further, it is covered on two-thirds of its surface by cartilage, which has not the regenerative power of periosteum.

Similar conditions prevail and probably account for non-union in intra-capsular (subcapital) fractures of the neck of the femur; and degenerative changes very occasionally occur, for the same reasons, in the intra-articular fragment of an ununited fracture of the proximal third of the carpal scaphoid.

The infrequent cases in which degenerative changes occur in the semilunar without any history of injury present a more difficult problem. Müller's suggestion that repeated minimal traumata incidental to any heavy occupation may suffice to cause the disease is borne out in the bilateral case in our series (Case 9) and by many other recorded cases.

It is further possible that the occasional examples of 'semilunitis' which may occur without injury, and are characterized by the early X-ray appearance of dense diffuse sclerosis which appears to indicate complete necrosis of the bone, may be due to an attenuated infection, as Axhausen suggests.

**Clinical Signs and Symptoms.**—These are remarkably constant, and fall into three periods, in the majority of cases, in which the lesion is caused by

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\* The separate ligament, which Delbet describes as the 'frein du semilunar', as, while intact, it prevents enucleation of the bone, appears to be merely the deeper fibres of the strong dorsal radiocarpal ligament, which runs obliquely downwards and inwards from the posterior border of the inferior articular surface of the radius, to become attached to the semilunar and cuneiform.

some definite injury. The injury commonly consists in a fall on the outstretched hand, or a blow on the base of the palm which causes forcible dorsiflexion, but occasionally the semilunar is damaged by a blow on the knuckles of the closed fist or on the back of the wrist. Pain, swelling, and limitation of the movements of the wrist ensue, and last from a few days up to two or three weeks. After a variable and often long quiescent interval, which lasts for months or years, some further slight injury, or perhaps continued occupational strain, determines a gradual return of the local symptoms, which eventually become sufficiently severe to compel the individual to cease work. Where there is no history of injury the onset of symptoms is insidious, and corresponds with this third stage, which is associated with definite deformation of the bone. The clinical manifestations are pain in the wrist, with maximum tenderness on palpation over the semilunar bone. Some oedematous swelling may be present over the back of the wrist, and in some cases a bony deformity, caused by projection of the flattened bone, may be appreciable. The hollow which can be felt with the tip of the finger in a normal wrist proximal to the head of the os magnum is then replaced by a bony prominence. Finsterer's signs may occasionally be present (*see* footnote, p. 579). Flexion and extension movements are invariably restricted to one-half or one-third of their normal range; and both active and passive movements are painful and occasionally accompanied by intra-articular crepitus. The relations of the styloid processes are unaltered. In a few cases some wasting of the muscles of the forearm has been noted. The latent period in the usual clinical history, and the gradual onset of the symptoms, exclude dislocation of the semilunar, while the X-ray findings—which alone permit of a correct diagnosis—differentiate the lesion under consideration from tubercle and from osteoarthritis of the carpus.

**X-ray Appearances.**—Skiagrams of the wrist show the bone to be flattened, and in lateral views it is seen to be sausage-shaped, the anterior and posterior horns being spread out over the head of the os magnum. The radial articular facet loses its convexity and becomes flattened. Its normally smooth outline is replaced by a wavy or irregular surface and the sharply defined limits of the normal bone are lost. Curved linear shadows are frequently present in the proximal third of the bone and are seen running more or less parallel to the radial articular surface. The bone markings are lost and islands of sclerosis with intervening areas of rarefaction appear.

Considerable stress has been laid upon the occurrence of fragmentation in the bone, but this is not a prominent feature in our cases. It is, however, present in *Case 6* and probably in *Case 1*.

In those cases where skiagrams of both wrists are available for comparison two additional features can be demonstrated. Thus in *Case 3* the semilunar appears to have rotated slightly round the head of the os magnum. *Cases 3* and *4* also show considerable shortening of the carpus on the diseased side.

In long-standing cases marked hypertrophic changes appear in the neighbouring bones, the styloid processes of the radius and ulna being chiefly affected.

**Histological Findings.**—These vary to some extent with the stage and duration of the lesion, but in general there is an association of active absorption

of the bone lamellæ together with replacement by granulation tissue which fills the enlarged bone spaces. This picture, which is characteristic of an early stage of bone repair, is in Kienböck's disease perpetuated by conversion of the granulation tissue into fibrous tissue, instead of calcification.

The microscopic appearances suggest that this is due to disturbance of the blood-supply, and the varying age of the fibrocellular tissue in different parts of the same section indicates that this fibrous replacement is a slow process which lasts for months and years. During this time, any fresh injury may serve to light up the local lesion, increasing the absorptive process and leading to deformation or pathological fracture.

**Prognosis and Treatment.**—Hosford's statement in a recent article that "the end-result of fractures of the scaphoid which have been overlooked tends to be a good deal worse than those which are recognized and treated immediately", is equally true of the semilunar, where, unfortunately, fractures are even less often diagnosed in their early stages.

If, however, a fissured fracture be recognized, the hand and wrist should be immobilized for six weeks on a short dorsiflexion splint, with the fingers flexed, before massage and active movements are commenced.

In the case of a compression fracture with gross deformity, and in the later stages of Kienböck's disease, where the semilunar is deformed and has lost all semblance of its normal shape, excision should be carried out as soon as possible, before osteo-arthritic changes develop in the contact articular surfaces, if the patient's age and occupation justify it.

In the early stages of Kienböck's disease, where the radiograms show sclerosis or 'mottling' without any, or with but little, alteration in outline, conservative treatment by rest and physiotherapy may still achieve a cure; but, with deformation of the bone, permanent disability with much discomfort and serious loss of function is inevitable, for, although some symptomatic relief may be effected by rest and non-operative methods of treatment, the troubles recur as soon as the patient attempts to resume labouring work.

**Operative Treatment: Excision of the Semilunar Bone.**—The dorsal route is generally employed, as the volar approach necessitates partial division of the anterior annular ligament, opening up of the common flexor sheath, and retraction of the median nerve.

*With the hand forcibly adducted at the wrist*, a straight incision of about two inches in length is made, with its proximal third over the lower end of the radius, just to the inner or ulnar side of the tubercle which marks the oblique groove for the long extensor of the thumb, whilst the distal two-thirds run down the back of the carpus in the depression—which can be felt by the finger-tip—between the tendon of the extensor carpi radialis brevis, on the outer side, and the extensor tendons of the index on the ulnar side (*Fig. 458*).

The posterior annular ligament is partly divided, and the common extensor tendons are retracted as far as possible inwards, and, *with the hand still held in forcible adduction*, the articulation is opened as far inwards under the extensor tendons as possible, because the semilunar lies under cover of them, and its outer side only is exposed on their radial side when the hand is fully adducted at the wrist.

The articulation between the scaphoid and semilunar is exposed when the edges of the incision in the posterior ligament are retracted, and with a sharp rugine the dorsal ligament which connects the semilunar with the os magnum below, and the lower edge of the radius and the posterior edge of the triangular fibrocartilage above, is gradually divided. A blunt-pointed tenotome is then slipped from below upwards into the articulations, with the scaphoid on the outer and the cuneiform on the inner side, to divide the interosseous ligaments between their proximal edges, and the semilunar is removed with a curved dental elevator or similar instrument. The friable bone invariably breaks up, and its complete removal is by no means an easy task, as the exposure is limited and tiny fragments are apt to be left adherent to the anterior ligament in the depths of the wound.

The removal should be careful and complete, as any residue may impair the post-operative result—e.g., *Case 3*.

The results after operation have been dealt with at some length in the after-histories of our own and other published cases. In general, the pain is relieved or cured, and the outlook as regards recovery of function is excellent, where progress is not impeded by any question of litigation. There is, however, some permanent loss of range of movement, which as a rule gives rise to insufficient disability to affect the wage-earning capacity in ordinary labouring work.

The *question of compensation* often arises in this disease. As we have seen, the lesion is certainly due to injury in the great majority of cases, and there is no doubt about the question of liability when the radiograms show a fissured or compression fracture subsequent to a definite injury. The difficulty arises in those cases in which the original injury was of a comparatively trivial nature, as a claim is seldom made at that time or during the short period of partial temporary disability which ensues. The quiescent interval may last for months or years, and compensation is usually claimed when the disability recurs, either as a result of some fresh injury or from the strain of heavy occupation. The radiograms then reveal the presence of an old-standing lesion which could not have been caused, but might have been aggravated, by recent injury, and if the occurrence of such can be proved, the employer at the time it was sustained may be held liable. When, however, the recurrent disability is due to occupational strain without any fresh accident, the liability should date back to the primary injury: but the lapse of time, and often the absence of notice, may prejudice any claim for compensation.

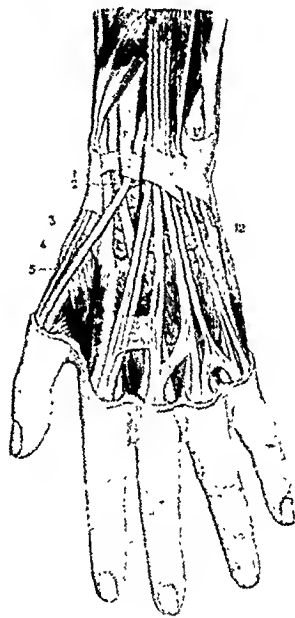


FIG. 458.—Line of incision just to the radial side of extensor tendons of index. 1, Extensor carpi radialis longior; 2, Extensor carpi radialis brevior; 3, Extensor ossis metacarpi pollicis; 4, Extensor brevis pollicis; 5, Extensor longus pollicis; Sc, Scaphoid; R, Radius; U, Ulna. (From Farabeuf, slightly modified.)

## SUMMARY.

Fracture of the carpal semilunar is not uncommon, though often unrecognized, and the resulting local reaction is treated as a sprain, without sufficient immobilization.

The process of repair in the damaged bone is retarded by the defective blood-supply and the poor reactive powers of its cartilaginous covering.

A slow progressive degeneration ('Kienböek's disease') with absorption of necrotic bone lamellæ and fibrous replacement then takes place during a varying and often long quiescent interval, which is ultimately followed by deformation of the bone, with recurrence of the local symptoms and continued disability.

A similar local reaction may result from repeated minimal occupational traumata, and in exceptional cases may be caused by an attenuated infection.

We are indebted to our colleagues Mr. Broekman and Mr. Lytle for *Cases* 1, 2, and 11, and to Mr. Lionel Sutcliffe of Chesterfield Hospital, and Dr. Grout, Radiologist to the Chesterfield and the Royal Hospital, Sheffield, for the histories and radiograms of *Cases* 8, 9, 10, and 12.

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## HYDATID PNEUMOTHORAX.

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APPROXIMATELY 10 per cent of the hydatid cysts occurring in the human body are found in the lungs. Many of these are located deep in the pulmonary substance, in the neighbourhood of the hilus, and before they are very large rupture into a main bronchus, thereby bringing about in quite 80 per cent of cases a spontaneous and lucky cure—a process of evacuation by paroxysmal expectoration ('la vomique hydatique curative' of Duvé<sup>1</sup>). Some persist, especially those peripherally located, and in the course of years grow to the size of an orange, an ostrich egg, or even larger, and bulge from the pleural envelope of the lung. These large cysts sooner or later undergo complications and cause serious troubles which call urgently for surgical relief. The complication of pneumothorax due to the rupture of a pulmonary cyst into the pleural cavity with coincident opening into a bronchus is decidedly uncommon.

Duvé, of Rouen, who writes learnedly and exhaustively on this as on most other aspects of echinococcus infection, has been able to collect records of not more than 41 cases up to January, 1931.<sup>2</sup> His treatise published in 1925,<sup>3</sup> based on an elaborate analysis of 32 authentic cases recorded, beginning with the classical ones of Mercier and Fouquier about a hundred years ago, and fortified by original experimental observations, is the best exposition extant of the subject of hydatid pneumothorax.

Since then but few additional cases have been recorded in medical literature. I might mention those described by Sewell<sup>4</sup> (quoted by Dew in his book, *Hydatid Disease*), Bacaloglu and Tanasesco,<sup>5</sup> Anderson,<sup>6</sup> Griffiths,<sup>7</sup> Villegas,<sup>8</sup> Hillemand,<sup>9</sup> and Pericie.<sup>10</sup> Duvé, however, wisely emphasizes the well-known fact that where hydatid disease is concerned diagnostic errors can easily arise, and no doubt many other true cases have been seen but have been misinterpreted. On the other hand, large pulmonary cysts containing air have sometimes been regarded erroneously as examples of pneumothorax. Occasionally, too, gaseous hepatic cysts rupture into the pleural cavity and produce symptoms and signs falsely suggestive of a hydatid pneumothorax of pulmonary origin.

The case now to be described is a true example of hydatid pneumothorax, with the added complication of secondary hydatid cysts growing from the pleura and in the lung. The primary cyst in the lung ruptured into the pleural cavity about eight years ago and was evacuated by operation, but unfortunately not until four months had elapsed from the date of rupture. The resulting fistula, seeing that a break-through into a bronchus had also occurred, is still running and occasionally discharges a small cyst. Other cysts or fragments of cysts are at rare intervals expectorated. The affected



lung, which had collapsed and had become crowded by air-pressure against the mediastinum, remained firmly adherent there and has never since expanded to any appreciable extent. Nevertheless the patient is at the present time wonderfully fit, all things considered, and in spite of occasional periods of disability continues to render valuable service to his country politically as well as industrially. For various reasons, therefore, I think this case, which was in the main under my surgical care in Dunedin, New Zealand, is worthy of being recorded in some detail.

### CASE REPORT.

T. B., a sheep farmer from boyhood, was thrown from his horse on Jan. 20, 1924. His age was then 47, and he had previously enjoyed the best of health. After lying dazed for a few moments he picked himself up, and with some difficulty remounted his horse, rode home, and went to bed. He slept but little that night, but in the morning he got up, and actually worked for an hour or more drafting sheep. He felt pain in the right shoulder and upper part of the right chest in front, and could only use his left arm in his work. He found himself becoming less and less fit for exertion, and on the fifth day after his accident consulted his doctor (the late and much esteemed Dr. Gabites, of Timaru).

On examination no external evidence of injury was discovered, but all the classical symptoms and signs of a right-sided hydropneumothorax were present. Fluid reached to the level of the 3rd costal cartilage: air filled the pleural cavity above this line; the heart apex was displaced an inch and a half to the left; heart-sounds were pure, but rhythm slightly irregular; the pulse-rate was 120 and the temperature 101° F.; breathing was rather distressed, 32 to the minute; and the face a little cyanosed; but there was no cough and there was no expectoration until quite a month had elapsed from the time of his injury. An X-ray examination confirmed the ordinary clinical findings, and the diagnosis arrived at was hydropneumothorax, resulting from laceration of the right lung. No abnormality was discovered in connection with the liver or kidneys or other organs.

With rest in bed and ordinary medical treatment the patient grew worse: breathing became more difficult, appetite was lost, and there was much windy distension of the abdomen. During the next three months the aspirating needle was used on five or six different occasions, some air at high pressure and slightly blood-stained fluid in quantity varying from 10 to 80 oz. being drawn off, and much though only temporary relief thereby obtained. Microscopic examination of this fluid showed only red cells, lymphocytes, and polymorphonuclear white cells. No tubercle bacilli were found.

After the last aspiration the patient felt well enough to return to his home and attempt a little work, but as soon as the fluid and air accumulated again he became cyanosed, weak, breathless, and feverish. He noted now that the little bouts of coughing with the expectoration of small quantities of thin watery straw-coloured salty fluid, which had been occurring for about two months, were getting more troublesome. Dr. Gabites had him X-rayed again without helpful result, and then brought him down to Dunedin for consultation, where he was placed in a private hospital under my care.

The case was clearly one of hydro- now verging on pyo-pneumothorax with marked displacement of the heart to the left, as if from high pressure of the air in the right pleural cavity. Naturally in a hydatid country, with a patient exposed all his life to infection from sheep-dogs, the possibility—nay, the probability—of a ruptured hydatid was envisaged, and a positive complement-fixation test of the patient's blood lent considerable support to this diagnosis.

FIRST OPERATION.—On May 29, 1924, four months after the accident, an extensive operation was performed. Ether was administered intratracheally, but this method proved of no special service as the right lung remained collapsed and adherent throughout.

A portion  $4\frac{1}{2}$  in. long of the eighth rib below the scapula was excised, and the right pleural cavity widely opened. I had a clear view inside and could insert the whole hand for exploration purposes (Fig. 459). The following conditions were noted: (1) Twenty ounces or more of thin yellowish fluid with numerous small daughter cysts the size of cherries and peas floating therein; (2) A soft tawny-coloured false-membrane like thick wet wash-leather coating the pleura, both parietal and visceral, and embedded in this coating countless numbers of little cysts the size of shot and barley grains; (3) A ruptured cyst the size of a large orange protruding through a jagged laceration in its adventitia, and located at the bottom of the compressed right lung.

The adventitia, easily torn, was opened up more widely and the mother membranes and numerous daughter cysts were evacuated. All the fluid and debris in

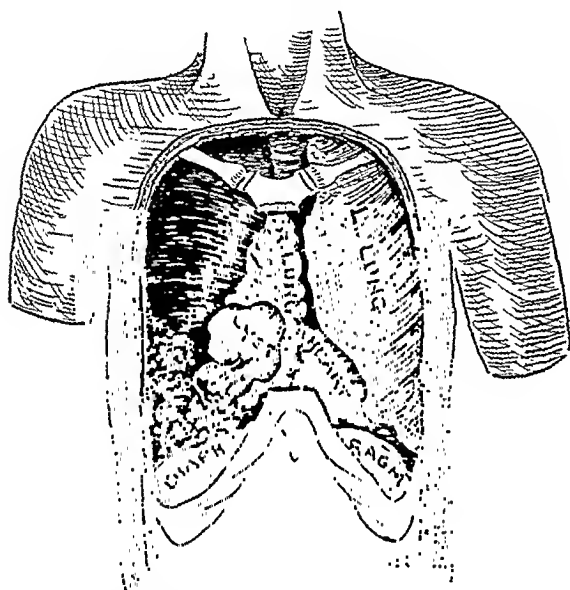


FIG. 459.—Diagrammatic sketch of the condition found at operation. The parasitic cyst is seen partly extruded through its torn adventitia at the bottom of the collapsed lung. Small secondary cysts are seen, some loose in the air-distended pleural cavity, some implanted in the thickened pleural coating.

the pleural cavity were flushed, sucked, and mopped out, and the pleural lining and the inside of the cavity in the lung were carefully wiped over with 2 per cent formalin solution. No bronchial fistula could be demonstrated and there was no expectoration by the patient during the operation. Nevertheless a bronchial fistula was present as subsequent events proved, and its valvular action favouring ingress and impeding egress was no doubt responsible for the accumulation of air under pressure in the right pleural cavity with consequent permanent collapse, compression displacement, and adherence of the right lung in the recesses of the mediastinum. Recognizing the probable existence of such a fistula I made an effort—unsuccessful as it turned out—to obliterate it, by puckering up and suturing the adventitia with chromicized catgut. The right lung was reduced to the size of an ordinary pear and the right pleural cavity was extraordinarily

large and empty. I could feel the vertebral column jutting into it as if the lung and mediastinal contents had been crowded out of their proper habitat over to the left side and had become solidly fixed there.

I closed the wound and made a futile attempt to dispense with drainage, but a week later had to put in a tube owing to a recurrence of the former symptoms of embarrassed respiration and fever.

Bacterial examination of the fluid evacuated at the time of operation showed Gram-negative bacilli—no tubercle bacilli—and, on culture, a profuse growth of coliform bacilli which gave the sugar reaction of *B. coli*.

After recovering sufficiently from this operation, the patient returned home wearing a tube, which was removed and re-inserted, and antiseptic irrigations were administered through it from time to time as occasion required.

For a year this state of affairs persisted and the patient's health was sometimes better, sometimes worse. Occasionally a small cyst and fragments of membrane would be discharged from the sinus, and occasionally also similar material would be expectorated.

SECOND OPERATION.—On Dec. 17, 1925, owing to faulty drainage, his condition was so unsatisfactory that another operation was performed by Dr. Gabites. Two

inches of two ribs in the vicinity of the sinus were removed, the pleural cavity again cleansed, some small cysts evacuated, and a larger tube inserted.

**THIRD OPERATION.**—With the death of Dr. Gabites, the patient came under the devoted and skilful care of Dr. W. H. Unwin, of Timaru, and he found it necessary on Jan. 5, 1926, to open once more into the pleural cavity. He made a very free incision, which permitted him to explore thoroughly. The lung remained totally collapsed, but on probing into it in the region of the interlobar sulcus, he found and evacuated a nest of small cysts—the result, no doubt, of implantation of scolices at the time of the original rupture of the cyst in January, 1924. Again the patient made a good recovery, and as before gained weight and strength and managed to do a fair amount of bodily and mental work. At intervals of a few months, however, trouble with the tube, poor drainage, sepsis, discharge of cysts by the fistula or by mouth and so on would recur in greater or less degree and incapacitate him for a time.

**SUBSEQUENT HISTORY.**—Thus he has gone on for several years, very pluckily making the best of things, and on the whole living a useful life. In 1929 he discharged two or three cysts of considerable size from the fistula and has been much better since.

Dr. Unwin wrote as follows on Feb. 8, 1931: "On the whole the patient has been well since May, 1929, and he is now 14 stone in weight. He gets occasional rises of temperature, usually the herald of the appearance of cysts in the discharge. The right pleural cavity has progressively decreased as shown by X-ray plates. About November last he informed me that a swelling appeared in the neighbourhood of the right breast, which went down rather quickly a day or two later, and a large cyst then appeared in the discharge. I did not see the swelling, but if the observation is correct it would seem that there is some infection in the chest wall. The complement-fixation and Casoni reactions are still strongly positive. If at a later date there is reason to believe that the patient has overcome his hydatid infection, an attempt might be made to close the cavity by some plastic procedure, combined if need be with avulsion of the phrenic nerve."

### HYDATID PNEUMOTHORAX IN GENERAL.

In a typical case, the patient is a young adult—more often a man than a woman—whose occupation has brought him into close association with country dogs, and who has thereby become infected with one or more hydatid cysts as a result of swallowing ova of the *Tenia echinococcus*, and the stage is set and the subsequent course of events takes place in the following way:—

1. A univesicular cyst, as large, say, as an orange, bulges subpleurally from the lower part of the right lung (*Fig. 460*). Its adventitia, composed of a compressed and fibrosed layer of lung and pleural tissue, is particularly thin and lacerable towards the chest wall, and, as is usual in univesicular, non-suppurating cysts, there are no protective pleural adhesions. Where the cyst abuts on the deeper regions of the lung towards the hilus, some of the larger bronchi are incorporated in the adventitia. The bronchial wall facing the cyst may be in places completely destroyed by a process of pressure-absorption exercised by the expanding cyst, but the lumen of the bronchus, owing to its epithelial lining, does not undergo organic occlusion. There are, indeed, one or more potential holes in these bronchi sealed only by the close coaptation of the hydatid ectocyst. (Cf. the analogous effect on bile-duets in the case of hepatic cysts.)

The blood-vessels of the adventitia, not being lined by epithelium, are mostly obliterated by pressure-fibrosis, but here and there a vessel of some size, capable of giving rise to considerable hæmorrhage, may persist.

The membranes of the parasitic cyst, consisting of the thick hyaline protective ectocyst lined by the thin germinal endocyst, are tightly pressed against the adventitia by reason of the high tension of the enclosed hydatid fluid. There is no organic union between the parasite and its host—close co-aptation suffices for the transference of nutriment by osmosis. The endocyst is dotted over with tiny brood-capsules just visible to the naked eye, and in these brood-capsules and distributed also in the clear watery fluid are myriads of microscopic scolices. The fluid and the scolices possess specific toxic properties.

2. The cyst ruptures (*Fig. 461*). This event may result from an accident, even a slight accident, or from a violent respiratory effort such as coughing or sneezing, from muscular violence, or it may arise spontaneously. It is obvious that a cyst

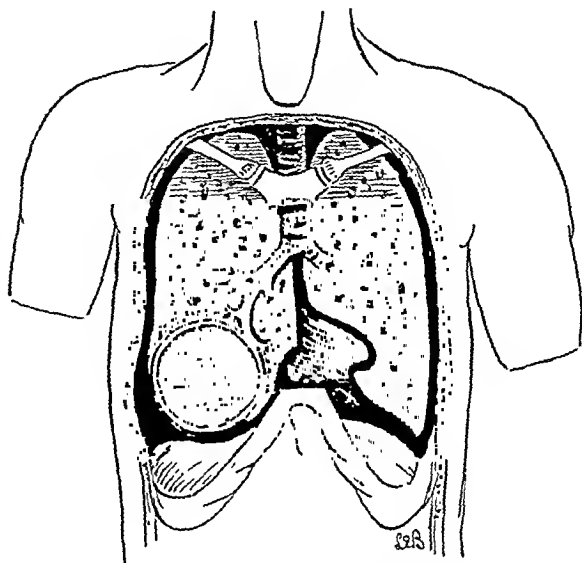


FIG. 460.—Diagram of condition before rupture of cyst. Note the cyst bulging subpleurally towards the chest wall and very thinly protected there. Note also the close relation of a deep bronchus to the cyst wall.

cannot go on enlarging indefinitely, and rupture is a common ending to the enlargement of a pulmonary cyst.

The thin adventitia on the pleural aspect of the cyst is torn open, and the parasitic membranes are similarly lacerated. Hydatid fluid is forced out into the pleural cavity, and coincidentally with this evacuation of fluid the mother membranes crumple and fall away from the adventitia. Thus a deficiency or actual hole is laid open in the wall of one or more of the larger bronchial tubes incorporated in the adventitia. Through this hole some fluid hydatid debris and perhaps some blood may escape into the air-passages and be coughed up paroxysmally,

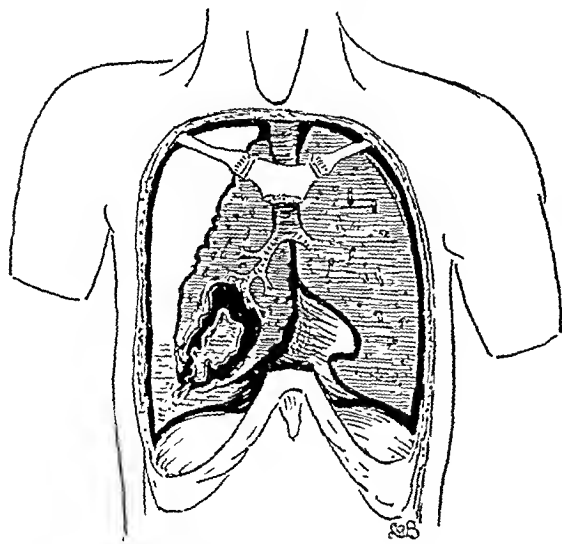


FIG. 461.—Diagram of condition immediately after rupture of cyst. The walls of the adventitia and of the parasitic cyst are lacerated; hydatid fluid and scolices escape into the pleural cavity, the parasitic cyst falls away from its adventitia exposing a hole in a bronchus; air enters the pleural cavity; and the lung collapses.

and conversely some air, some bronchial mucus, and possibly some bronchial bacteria, may be drawn by inspiratory action into the cyst cavity, and thence into the pleural cavity.

It sometimes happens, as in the case described above, that, owing maybe to the shape of the hole in the bronchus (*en bec de flûte*), or to a flapping tag of bronchial wall, or to a partly adherent plug of necrosed material, or to the crumpled mass of mother membrane, or to the irregular accordion-like collapse of the thin adventitia, that a valvular mechanism is introduced, permitting air to enter into the pleural cavity but preventing its escape again by way of the perforated bronchus.

3. Thus arises a pneumothorax of high pressure and in consequence the collapsed lung is further compressed and crowded against and even beyond

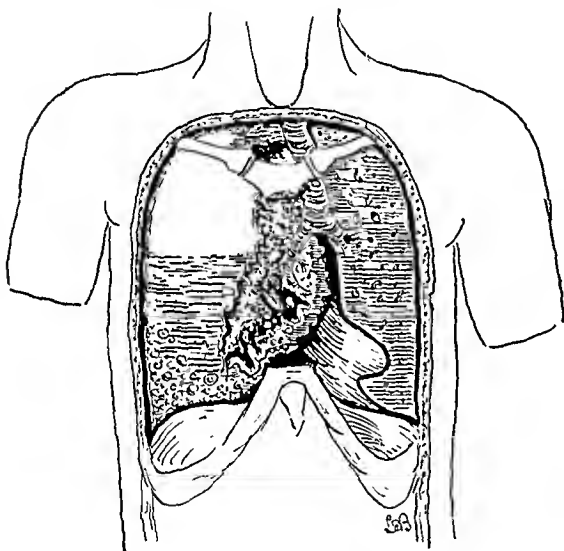
the mediastinum (*Fig. 462*). If this displacement persists for many days, the lung becomes adherent—perhaps permanently adherent—in its abnormal location. The heart also is pushed out of position; the main veins, particularly the inferior vena cava, are kinked; and the circulation of blood through them seriously impeded. Thus one can account for the breathlessness, cyanosis, and cardiac embarrassment that are characteristic of this type of pneumothorax.

4. In addition to the accumulation of air, hydatid fluid, fragments of membrane, scolices, a little bronchial secretion, possibly infected, and some blood escape into the affected pleural cavity. The outpouring into the pleural cavity of the toxic contents of the hydatid cyst may produce:—

*a.* Sudden pain and shock, followed by anaphylactic symptoms (inconstant in nature and degree) such as pruritus, urticaria, fever, vomiting, delirium, collapse, etc.

*b.* A defensive reaction in the pleura with exudation of pleuritic fluid and the formation of a fibrino-cellular false membrane coating the lung and walls of the pleural cavity.

*c.* A secondary echinococcosis developing in the pleura and in the pulmonary tissue of the cavity formerly occupied by the parent cyst. This secondary development of cysts, which takes some weeks or months to manifest itself, is due to the dissemination of scolices at the time of rupture



*Fig. 462.*—Diagram of condition in later stages after removal by operation of the primary cyst. The walls of the cyst cavity are crumpled together, but the bronchial fistula persists. Secondary cysts have formed in the lung and pleura, where dissemination of scolices had previously occurred. The collapsed and compressed lung, the trachea, and the heart have been pushed over towards the left side. The right pleural cavity contains air and pus.

of the fertile parent cyst, and their subsequent growth. As a surgical curiosity it may be mentioned that the whole parent cyst intact has escaped into the pleural cavity and continued to grow there (Mercier,<sup>11</sup> Dévé<sup>12, 13</sup>).

d. Lastly, a septic infection is sooner or later introduced into so favourable a medium by contaminated mucus entering by way of the bronchial aperture, and the hydropneumothorax becomes a pyopneumothorax. The bacteria concerned are usually pneumococci, staphylococci, coli bacilli, and occasionally gas-producing anaerobic organisms are superadded.

### DIAGNOSIS OF HYDATID PNEUMOTHORAX.

There is no need in this paper to detail the classical symptoms and signs of ordinary hydro- or pyopneumothorax—the tympanic resonance, the shifting dullness, the metallic tinkling, and what not—nor to describe the X-ray appearances. Suffice it to say, that there is no difficulty in diagnosing that a hydro- or a pyopneumothorax is present. Moreover, we should have no difficulty in diagnosing the hydatid factor *if only we keep in mind its possibility*. Hitherto the timely recognition of this hydatid factor has been missed more often than not, and the true pathology of the hydatid pneumothorax has not been revealed until the patient has actually coughed up his own diagnostic label, as it were, or the practitioner finds it at operation or post-mortem.

Early diagnosis is essential to successful treatment. Delay is disastrous, and therefore whenever a case of pneumothorax is encountered, particularly in a hydatid country, the possibility of hydatid origin should be taken into consideration. We must not make the common mistake of our predecessors and conclude that if a pneumothorax is not traumatic it must be tuberculous in origin. The tuberculous type is, of course, the commonest and it is usually correctly diagnosed; its clinical features, its bacteriology, its X-ray findings are all so manifest. In the hydatid type, except in the rare instances where tuberculous disease is combined, the usual tuberculous evidence is lacking. The case therefore should strike us as out of the ordinary. Hydatid disease should be thought of and the aid of the laboratory invoked, for of late years the laboratory technique in this connection has made such marvellous progress that a correct diagnosis of hydatid infection is rendered possible in fully 90 per cent of cases submitted. The tests most commonly and successfully applied are the skin reaction of Casoni and the blood complement deviation test.

### TREATMENT.

Needless to say, the sooner the patient is operated on the better. If immediately after the catastrophic rupture of the cyst, or at any rate within a few days of it, the thorax on the affected side is freely opened in a dependent position, the ruptured parasitic cyst and its contents completely evacuated, the pleural surfaces and the walls of cavity formerly occupied by the cyst cautiously wiped over with ether or with 2 per cent solution of formalin in water, and the pleural and cyst cavities drained for a few days, the case is likely to do well. The cyst cavity obliterates, the bronchial fistula closes, the lung re-expands, the wound heals, and the patient may be completely and permanently cured.

If the bronchial fistula does not close, there will obviously be interference with the expansion of the lung and with the healing of the wound, with resulting chronic empyema. It may then be necessary to make efforts to close the bronchial opening by inserting puckering sutures of chromicized catgut in the freely exposed affected portion of the lung—also to disinfect the empyema cavity with Dakin's or some such antiseptic lotion—and eventually, if need be, resort to one or other of the recognized plastic operations recommended for the obliteration of a chronic thoracic empyema—extensive rib resections, decortication, and so on.

But suppose it happens, as in the case here described, that the ruptured hydatid cyst is not promptly diagnosed and operated on, then, in addition to chronic collapse and adhesion of the lung and probable persistence of a bronchial fistula, a further complication arises that adds enormously to the difficulties of successful treatment—namely, the development of secondary cysts as the result of the implantation and growth of some of the innumerable scolices disseminated at the time of rupture of the parent cyst. From time to time some of these secondary cysts break away from their moorings and escape, either by way of the air-passages in the form of hydatid expectoration, or by way of the external opening in the thoracic wall.

Such difficulties in the way of successful treatment, as well as those of the usual chronic septic infection, are well exemplified in the case recorded, and the paramount importance of early diagnosis cannot be too strongly emphasized.

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## ON SOME COMPLICATIONS OF TRAUMATIC DISLOCATION OF THE HIP-JOINT.

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It is interesting to contemplate the generous space allotted to the description of the various types of traumatic dislocation of the hip-joint in the surgical text-books of a bygone age. There is no reason to suppose that this injury, which in modern surgical practice is completely overshadowed by fractures of the femoral neck, was more common in the epoch which inspired the classical monograph of Bigelow. To-day, the arrival in a hospital casualty department of an individual with a dislocated hip creates a thrill of excitement amongst the resident staff. The average dislocation is reduced without special difficulty, and the patient leaves hospital after a brief stay, rarely to be seen again. This course of events suggests what we know to be true: that following an uncomplicated dislocation of the hip-joint, the restoration of perfect function is the rule. But in certain circumstances the prognosis may be profoundly modified by the occurrence of complications which usually date from the time of the accident, but on occasion may arise during the early stages of treatment. The most important of these are: (1) Fractures of the acetabulum, or head of the femur; and (2) Involvement of the sciatic nerve. The significance of such complicated injuries is illustrated in the following cases which have come under observation during the past ten years.

### CASE REPORTS.

**Case 1.**—Posterior dislocation of the right hip. Fracture of acetabular rim. Extrusion of loose bony fragment per vaginam.

**HISTORY.**—The patient, an active woman of 30, sustained a dislocation of the right hip during a fall downstairs. The condition was diagnosed shortly after the accident by her doctor, who reduced the dislocation without difficulty, but found that the displacement of the head of the femur recurred almost immediately. This happened on several occasions, and each time reduction was effected.

**ON EXAMINATION** (Dec. 13, 1921).—When seen by the writer three weeks after the accident, the hip showed the signs of a stable reduction, but was sensitive on movement. In view of the unusual history, a short period of fixation in a plaster-of-Paris spica was advised. (For various reasons a radiographic examination could not be conveniently carried out.)

**SUBSEQUENT PROGRESS.**—The joint became comfortable when securely immobilized, and following the removal of the plaster some three weeks later, a full range of painless mobility was rapidly regained. Eight weeks from the date of the accident a semicircular fragment of bone was passed per vaginam during a menstrual period. Unfortunately the fragment was lost, but from the description given by the patient, there was little doubt that it represented a portion of the acetabulum—most probably the rim. On further inquiry the patient stated that her menstrual periods since the accident had been accompanied by severe pain—an unusual



occurrence—and that after the extrusion of the bony fragment the pain had disappeared completely.

**RESULT.**—The patient showed no trace of hip-joint disability ten years from the time of the accident.

**Comments.**—In the absence of any radiographic evidence, the exact site of origin of the detached fragment could not be established. The clinical sequence of events suggests that the dislocation was accompanied by a fracture of the acetabular lip, with displacement of a fragment which at first formed an obstacle to concentric reposition of the femoral head, and rendered the reduction temporarily unstable. Some days later the fragment penetrated the vaginal wall, and by a gradual process of ulceration was finally extruded into the vaginal cavity.

**Case 2.**—Posterior dislocation of the right hip. Sciatic nerve involvement. Myositis ossificans.

**HISTORY.**—The patient was a man of 50, suffering from temporary mental derangement, who jumped through the window of a railway carriage, and sustained a number of injuries. After three weeks' treatment in hospital he was removed to his home, where an unreduced posterior dislocation of the right hip was discovered: at this time there was also evidence of a severe sciatic nerve lesion. Three separate attempts were made under anaesthesia to reduce the hip by manipulation, the third attempt being successful. After many weeks' treatment of the nerve injury by the usual physiotherapeutic measures, the patient began to bear weight on the injured limb, using a walking appliance to control the foot-drop. The hip-joint remained stiff, and there was very little sign of recovery in the function of the sciatic nerve.

**ON EXAMINATION** (June 30, 1924).—When seen by the writer nine months from the time of the accident, the patient was walking with considerable difficulty. The right hip was almost completely fixed in a position of slight flexion and negative abduction. The sciatic nerve function showed a complete motor block with marked wasting of both the anterior and posterior tibial muscular groups: the sensory block was not absolutely complete. There were no trophic complications. An X-ray (*Fig. 463*) of the injured hip showed: (1) The femoral head correctly placed in the acetabulum, with a fairly clear joint space; (2) Large masses of new bone surrounding the joint, especially on the lateral aspect. In view of the severe sciatic lesion, exploration of the nerve-trunk was advised,



**FIG. 463.**—Shows large masses of bone surrounding the hip-joint.

and at the same time it was proposed to make an attempt to mobilize the hip-joint.

**OPERATION** (July 28, 1924).—(1) *Neurolysis of the sciatic nerve*; (2) *Excision of intramuscular and capsular new bone*; (3) *Excision of head of femur*.

1. The hip-joint was exposed by the postero-lateral route, after detachment of the great trochanter. The pelvi-trochanteric muscles and the joint capsule were found to be extensively invaded by masses of bony tissue (*Fig. 464*). After a free

excision of the bony plaques, the joint remained fixed, owing to the close fibrous connection which existed between the femoral head and the socket. It was therefore decided to construct a pseudarthrosis by excision of the femoral head.

2. The sciatic nerve was now exposed. The nerve was found to be intact, and running across a bony bridge behind the femur. Numerous bony spicules were adherent to the nerve-sheath, which on the lateral aspect especially was thickened and adherent over a considerable area. Direct faradic stimulation induced a response in the internal popliteal muscles, but no response in any muscle of the external popliteal group. The nerve-trunk was now freed, all adherent bony tissue being dissected away. In order to provide a suitable bed, a pedicled flap from the deeper aspect of the gluteus maximus was interposed between the nerve and the bony pelvis.

3. The trochanter was reattached to the femur at a lower level in accordance with the technique of reconstruction of the hip-joint. After closure of the wound, traction was applied to the limb with the hip in moderate abduction.

**SUBSEQUENT PROGRESS.**—Six months later the reconstructed hip showed a moderate range of passive mobility, but the active muscular control over the joint was relatively poor. The foot-drop was still present, and there was no evidence of motor or sensory recovery in the external popliteal distribution. There was, however, a definite return of power in the posterior tibial group, and the recovery was complicated by the appearance of the signs of severe irritation—namely, pain in the foot and trophic ulceration. These symptoms were so distressing that the patient begged to have the limb amputated. A further period of conservative treatment was recommended. The ultimate fate of the patient is unknown.

**Comments.**—In this case the exact chronology of the sciatic nerve complication is uncertain, but whether there was a primary contusion or not, the nerve sustained additional damage by its late involvement in fibro-osseous scar. The appearance of irritation signs in the stage of recovery is, of course, a typical phenomenon in severe but incomplete sciatic lesions. The intramuscular and capsular ossification points to the dangers of attempting reduction by manipulation in a dislocation of some weeks' standing.

**Case 3.**—Posterior dislocation of the left hip. Fracture of head of femur. Fracture of acetabular margin. Sciatic nerve involvement.

**HISTORY.**—The patient was a woman of 60, who was injured in a motor accident in which she sustained: (1) minor superficial skin wounds, (2) slight concussion, and (3) a posterior dislocation of the left hip. The dislocation was recognized promptly, and reduced on the day of the accident. Almost immediately afterwards the signs of a complete sciatic lesion were discovered in the same limb. A radiogram of the hip showed a fracture of the femoral head with detachment of a comparatively large fragment. The usual mechanical and physiotherapeutic treatment for the nerve injury was instituted, and a few weeks later signs of



FIG. 464.—Bony masses removed from pelvis-trochanteric muscles and joint capsule.

recovery were noted in the internal popliteal distribution. During this time the patient was able to move the hip freely whilst lying in bed.

ON EXAMINATION.—When seen by the writer seven weeks after the accident, the left hip showed an almost complete range of mobility without discomfort. All the signs of a severe sciatic lesion were present: a complete motor and sensory block in the external popliteal distribution, and a severe but incomplete block in the internal popliteal distribution. Considerable pain was experienced in the foot, but the trophic condition was excellent.

An X-ray (*Fig. 465*) of the left hip showed: (1) A large fragment detached from the femoral head and lying on the outer aspect of the joint; (2) A thin strip of bone lying between the loose capital fragment and the acetabular margin. It was decided to treat the nerve injury conservatively for a further period. During



*FIG. 465.*—Shows: (1) Large fragment detached from femoral head; (2) Small fragment detached from acetabular margin. Both fragments lie within the joint capsule.

the next four weeks there was additional evidence of motor recovery in the internal popliteal distribution, but the irritative symptoms became worse. For this reason exploration of the sciatic nerve was recommended.

OPERATION (Oct. 28, 1930).—(1) *Neuralysis of the sciatic nerve*; (2) *Removal of bony fragments from the hip-joint.*

1. The sciatic nerve was exposed from the brim of the pelvis to the lower border of the gluteus maximus. The nerve-trunk was intact, but much flattened, with a thickened sheath bound down by tough adhesions over the whole of this area. As it passed over the region of the joint capsule the nerve showed a definite constriction, and at this level was closely adherent to the short external rotator muscles. Faradic stimulation elicited no response in the external popliteal muscles, but a feeble twitch was noted in the tibialis posterior. The nerve-trunk was now completely freed, and preparations were made to isolate it from the scar tissue in its former bed. Before completing this part of the operation, it was decided to inspect the hip-joint more closely.

2. The tendon of the obturator externus was divided and the joint capsule exposed. A definite irregularity could be felt in the underlying femoral head. The capsule was accordingly opened by a short incision, and the large loose bony fragment shown in *Fig. 465* was removed. This fragment showed all the appearances of 'aseptic necrosis'. The second, more slender fragment, which was adherent to the

joint capsule, was also removed. The capsular incision was closed and the divided obturator externus tendon sutured.

3. The sciatic nerve was shut off from its former bed by means of a pedicled flap from the deeper fibres of the gluteus maximus.

RESULT.—One year since the operation. The patient is walking with fair comfort, and the hip-joint shows a complete range of painless mobility. There is still evidence of a fairly severe sciatic lesion, although considerable recovery has occurred in the posterior tibial group. Slight return of power is also apparent in the following muscles of the tibial group: tibialis anticus and extensor longus digitorum.

Comments.—This case illustrates the typical sciatic lesion accompanying a dislocation of the hip-joint, i.e., a primary contusion of the nerve-trunk

which later becomes adherent to the structures on the posterior aspect of the femoral head and neck. The irritation symptoms are also characteristic. The fracture of the femoral head is an example of an unusual complication, and the rapid restoration of excellent joint function, despite the presence of a large loose fragment, is most striking.

*Case 4.*—Traumatic dislocation of the right hip. Fracture of acetabular margin.

**HISTORY.**—The patient, a man aged 27, was injured in a motor accident in Belgium. He was admitted to hospital temporarily unconscious, and was there discovered to have a dislocation of the right hip. The dislocation was reduced on the day of the accident, and nine days later he was allowed to walk about. The hip continued to be stiff and painful.

**ON EXAMINATION** (June 18, 1931).—When seen by the writer nineteen days after the accident, the patient limped badly and bore very little weight on the injured limb. The right hip showed a definite limitation of abduction, flexion, and internal rotation, with considerable pain and muscular spasm when movements were forced. The limb appeared to be half an inch longer than its fellow.

An X-ray (*Fig. 466*) showed: (1) An irregularity of the acetabular margin, with the displacement of a considerable fragment into the joint, the fragment lying between the femoral head and the socket; (2) A bony 'spike' just above the lesser trochanter. This was regarded as an anomaly of long-standing. In view of the signs of intra-articular block an open operation was advised.

**OPERATION** (June 25, 1931).—*Arthrotomy; removal of bony fragment from acetabulum.*

The hip-joint was exposed by a lateral approach in the interval between the tensor fasciæ femoris and gluteus medius. After inspecting the rim of the acetabulum on the upper and posterior aspects, it was considered advisable to redislocate the femoral head in order to obtain a full view of the interior of the socket. It was noted that the cartilaginous covering of the head was intact, and that the ligamentum teres had been torn completely across. In the depths of the acetabulum, lightly attached at one point, was a fair-sized bony fragment representing part of the fractured acetabular lip; this was removed, and a second much smaller fragment was discovered lying free in the socket; this was also removed. The femoral head was now replaced in the acetabulum, but no attempt was made to suture the very small incision in the joint capsule.

**SUBSEQUENT PROGRESS.**—Traction was applied to the limb in slight abduction. This was maintained for three weeks, and the patient was then allowed to move the joint freely in bed. Cautious weight-bearing was begun in six weeks from the time of the operation.

**RESULT.**—After six months the patient is walking without any limp, and the hip-joint shows perfect mobility without any trace of discomfort.

**Comments.**—In this case, as in *Case 1*, the detachment of a fragment from the acetabular lip produced an obstacle to the concentric reposition of the femoral head. Owing to the position of the fragment in the depths of the socket, removal by open operation was essential.



**FIG. 466.**—Irregularity of acetabular margin with displaced fragment lying between the femoral head and the socket.

## A CASE OF PARATHYROID TUMOUR ASSOCIATED WITH FIBROCYSTIC DISEASE.\*

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### WITH A PATHOLOGICAL ACCOUNT

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ALTHOUGH the metabolic investigations of this patient are unfortunately not as complete as might have been wished, the case nevertheless seems worthy of record because of certain unusual features and because of the abundance of pathological material obtained. Excellent summaries of the literature are now available, and there does not appear to be any occasion here for an extensive review.

### CLINICAL REPORT.

L. W. was a normally developed girl aged 20: there was nothing of importance in her family or personal history until September, 1928, when she began to feel pains in her left buttock and thigh. For this she was seen at two hospitals, but apparently no skiagrams were taken and no abnormality was discovered.

On Feb. 2, 1929, while the patient was sitting on the 'chamber', her left lower limb gave way and she fell to the ground. She was seen by Dr. Vane-Sutton, who found a *fracture of the left femur* and immediately transferred her to the Middlesex Hospital, where she was admitted under the care of one of us (G. G-T.). The limb was put up with weight extension from tibial screws after the method of Meurice Sinelair and good alinement was obtained. An X-ray now showed at the junction of the middle and upper thirds of the left femur an area of rarefied bone about six inches long, through the middle of which ran the line of fracture: the cortical bone was greatly narrowed, the central bone showed a mottled appearance; the upper and lower limits of the diseased area were sharply demarcated from the adjacent bone (*Fig. 467*). Radiograms of the pelvis, the upper part of the right femur, and the region of the left knee showed no abnormality, with the exception of a doubtful area in the right ischium. Accordingly a diagnosis was made of 'localized fibrocystic disease'.

Rather as a matter of general interest the serum calcium and phosphorus were estimated and were found to be 16.9 and 4.2 mgrm. per 100 c.c. during the first month after her admission. At this time we were unfortunately unaware of the parathyroid syndrome, which had hardly been discussed in the English literature, and no further steps were taken at that period in pursuing these metabolic investigations of the case.

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\* Received for publication Oct. 20, 1931.

By July, 1929, there was no evidence of union of the fracture, and the pain and tenderness in the left thigh and buttock were unabated. Skiagraphy showed the disease to be still demarcated abruptly, but within this zone the bony structure was now completely lost and definite tumour formation was discernible (*Fig. 468*). The rest of the femur showed a decrease in density, which at the time was interpreted as disuse atrophy. After consultation with colleagues the suggestion of a malignant neoplasm was mooted, and under these circumstances there remained no alternative but to remove the limb,



FIG. 467.

FIG. 468.

FIGS. 467, 468.—X-ray appearance of left femur on Feb. 2, 1929, and on July 3, 1929.

which was disarticulated at the hip-joint on July 4, 1929. On Aug. 2, the patient was discharged to a Convalescent Home; she subsequently returned to her own home walking on crutches.

In September, 1929, our attention was drawn to a case of parathyroid tumour under the care of Dr. Donald Hunter at the London Hospital, and consequently to other cases reported on the Continent; the patient was therefore summoned again to hospital that we might reconsider or revise the original

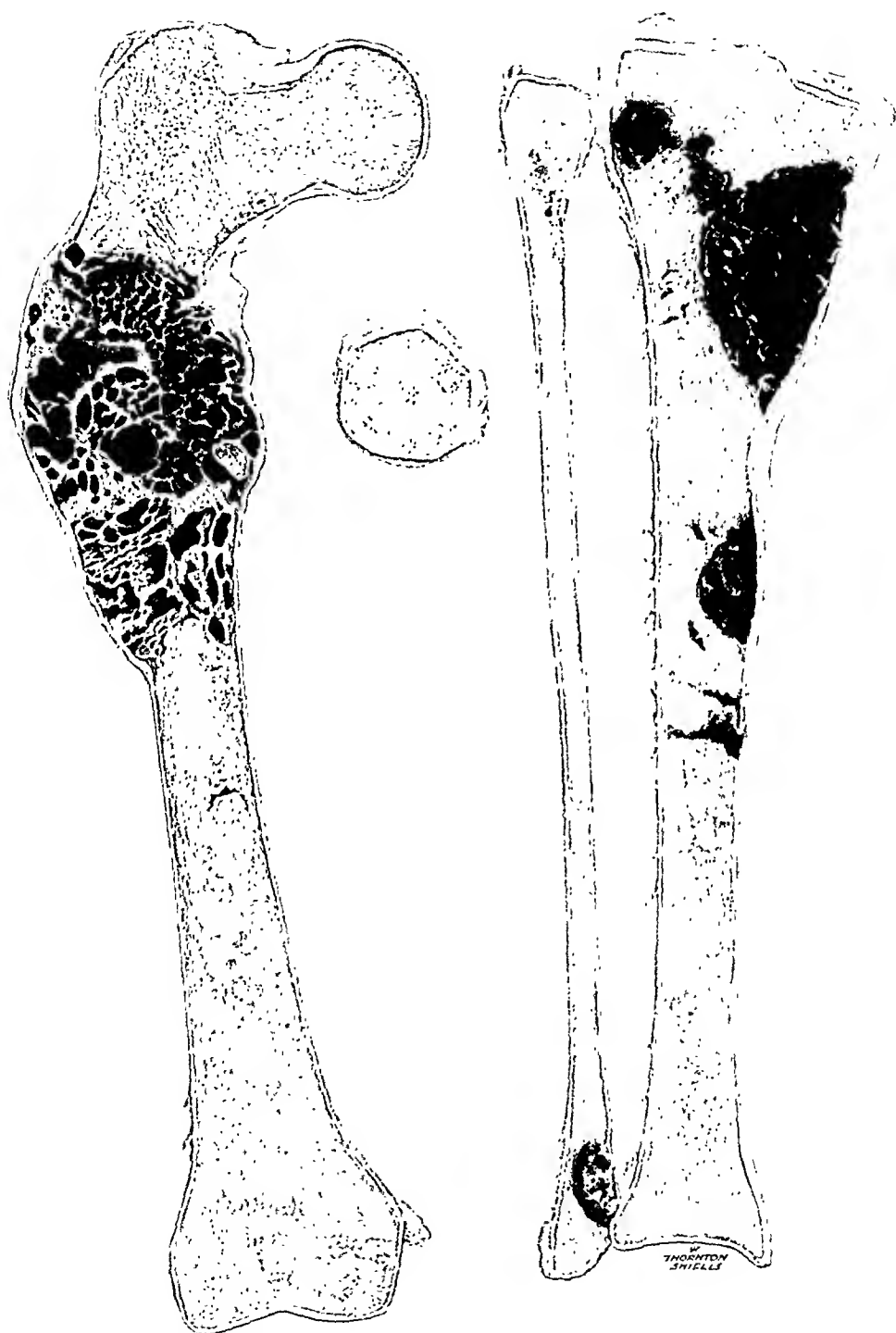


FIG. 471.—The femur, patella, tibia, and fibula. (For description, see text.)

the normal medullary structure. The latter is most marked in the metacarpals and phalanges, which were comparatively slightly affected in the first place and are now practically normal, thereby giving hope that in the fullness of time there may perhaps be a return of the whole skeleton to a normal structure.

### **PATHOLOGICAL ANATOMY.**

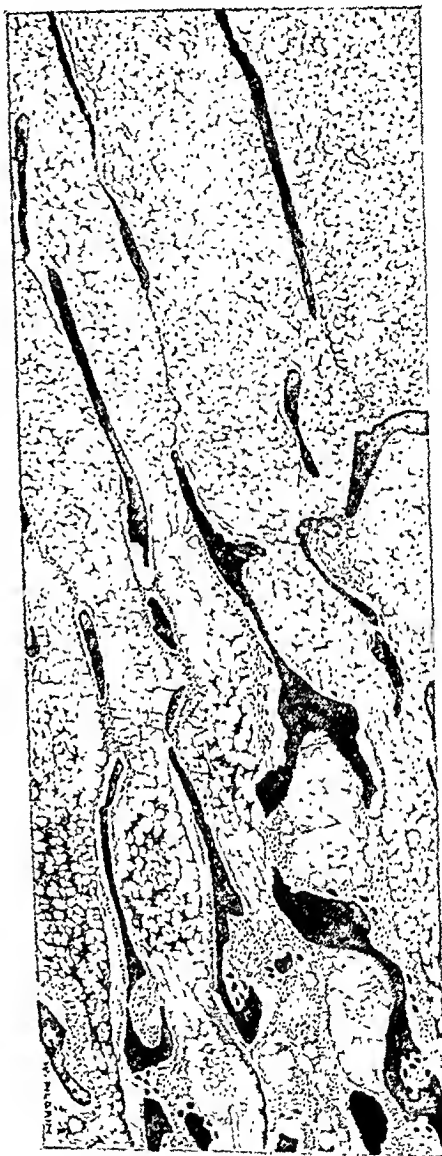
(PROFESSOR S. L. BAKER.)

1. **The Bones.**—The specimen consisted of the left femur, patella, tibia, and fibula. The upper part of the shaft of the femur at the site of the original fracture was distended by a large fusiform tumour-like mass which interrupted the continuity of the bone. All the bones in the specimen were found to be so soft that it was possible to cut the latter down with a knife. On bisection the appearances were as shown in *Fig. 471*.

In addition to the mass in the femur, there are several similar red cystic masses in the tibia, and in the lower end of the fibula and in the patella are smaller reddish areas without cysts.

Apart from these masses of red tissue, there is an extreme general atrophy of the bony structure. This is best appreciated by a close inspection of the lower part of the shafts of the femur and tibia, where there is no sign of red tissue, and where so little bone is present that the compact cortex is represented by only a few trabeculae beneath the periosteum; these can be squeezed in by moderate pressure between the finger and thumb. There is no visible replacement of structure by fibrous tissue, and there are no cysts other than those in the tumour-like masses.

**Microscopical Examination.**—The microscopic picture varies according to the site examined: a definite histology will be found to characterize the various areas of naked-eye pathological change. Sections were made of the following portions of the material: (1) Cancellous bone with pale fatty marrow; (2) Rather ill-defined streaks of pinkish tissue (seen best in *Fig. 471* in the cortical part

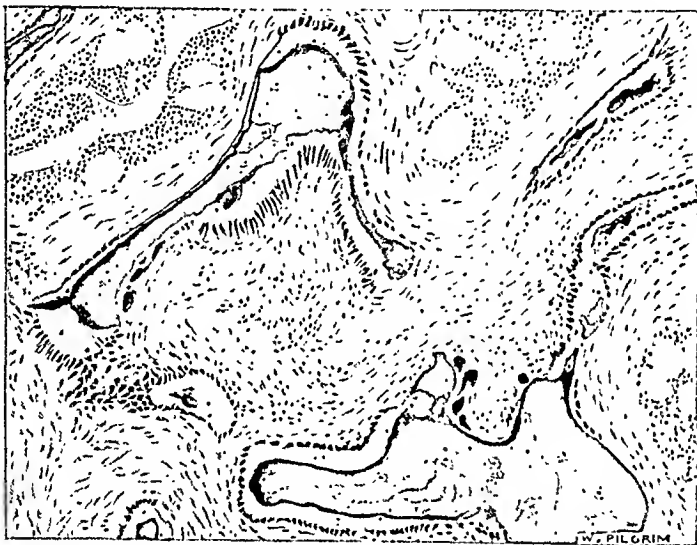


**FIG. 472.**—Low-power view of a section taken from the middle of the neck of the femur. This shows atrophic bony trabeculae and fatty marrow in the upper part: in the lower portion the trabeculae are clothed by spindle-cell tissue, and both osteoclastic destruction and new bone formation are in progress. There is also blood-forming marrow tissue lying between the fat cells.



of the shaft of the femur in its middle third below the site of the tumour); (3) Red cystic masses; and (4) Smaller reddish areas without cysts, such as that seen in the patella.

*Fig. 472* shows a low-power view (2-in. obj.) of a section taken from the central portion of the neck of the femur about one inch from the articular surface at a point where pale fatty marrow (1) gives place to an ill-defined pinkish tissue (this is hardly visible in *Fig. 471*). In the upper part of the figure are seen very thin but otherwise apparently normal bony trabeculae separated by fatty marrow. Towards the centre of the field the trabeculae become thicker and more irregular in shape, and each is surrounded by a sheath of spindle-cell tissue. Numerous osteoclasts can be seen in relation to the bony surfaces, some in groups situated in large irregular lacunae, such as are visible in the lower right-hand corner of *Fig. 472*. This lacunar



*FIG. 473.*—High-power view of the same region as that seen in the lower part of *Fig. 472*, showing the formation of new osteoid zones on the surface of calcified trabeculae. Lacunar resorption of bone at adjacent spots on the same trabeculae.

resorption of the bone is occurring here mainly on one side (right in *Fig. 472*) of the trabeculae; on the opposite side most of the trabeculae show newly-formed osteoid zones. In the marrow spaces there are also signs of activity, collections of blood-forming cells being interspersed among the fat globules. It is this red marrow formation which is mainly responsible for the pale-pinkish appearance visible to the naked eye.

*Fig. 473* shows, under a higher power, a few trabeculae in the same region as the lower part of *Fig. 472*. At this spot fresh osteoid tissue is being deposited on the surface of the older calcified (darker staining) bone through the agency of rows of osteoblasts, many of which appear columnar in shape, owing to the obliquity of the plane of section. At an adjacent spot on the same trabecula a line of osteoclasts lying in a shallow lacuna is engaged in the process of erosion. In the upper part of the figure the marrow

space shows a few oval fat cells surrounded by blood-forming marrow: in the remainder of the field the marrow space is occupied by a spindle-celled connective tissue.

The red cystic masses (3) are composed of spindle-celled connective tissue containing numerous multinucleated osteoclasts. Most of the cysts contain

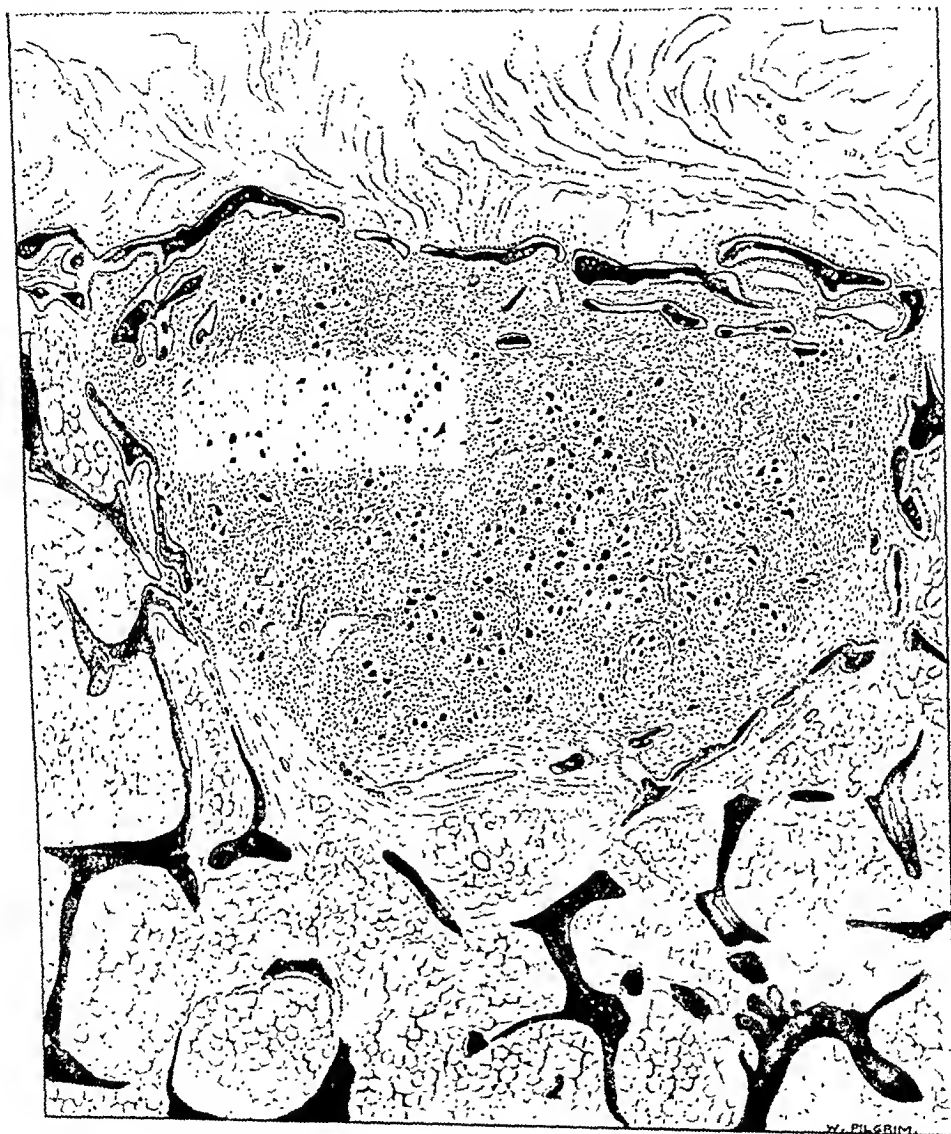


FIG. 474.—Low-power view of an area of pink tissue in the patella. Multinucleated osteoclasts (black spots) scattered about in spindle-cell tissue.

red blood-cells; some contain a clear fluid, possibly serum, which is seen in sections as a faintly eosinophil hyaline material. Trabeculae, of which some are calcified and others mainly osteoid, extend in an irregular and fragmentary manner into the spindle-cell tissue. Apart from the cyst formation and occasional foci of closely packed osteoclasts, the appearances are much the same as those seen in Fig. 474.

*Fig. 474* shows a low-power (2-in. obj.) drawing of a small reddish non-cystic area in the patella. At the top of the figure is the periosteal surface; under this is a rounded mass composed of spindle-celled connective tissue in which are lying numerous osteoclasts which appear as irregular black spots. Within this mass are no calcified bony trabeculae, but a few irregular strands and rounded masses of osteoid tissue. These show no definite lamellar structure and their fibres are irregularly arranged. Small areas of obviously coarse-fibred osteoid tissue are being formed in some parts of the spindle-celled matrix, but this feature is not well shown in *Fig. 474*. The field around this cellular mass is occupied by bony trabeculae which stain a deep slate-blue with iron hæmatoxylin and appear to be well calcified.

In short, the histological features of these bones exhibit a general atrophy of bony tissue, much of which appears to be in a quiescent state, with thin

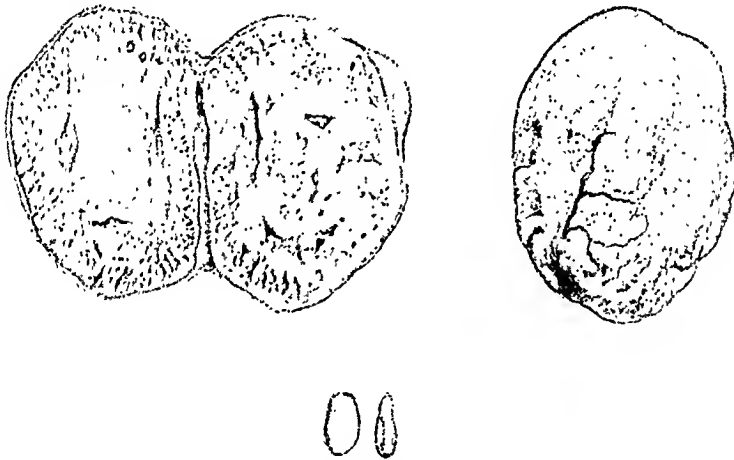


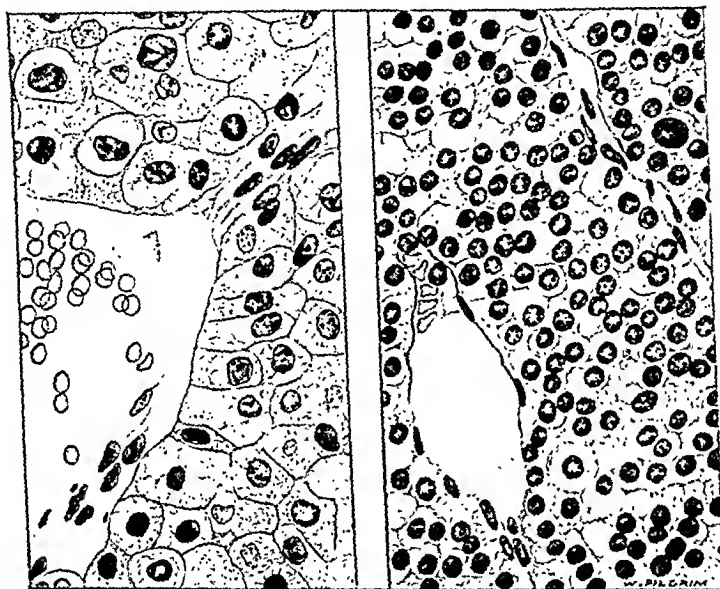
FIG. 475.—The parathyroid tumour, sectional and external views. Below, an apparently normal parathyroid removed at the same time.

trabeculae separated by fatty marrow. In places an active process is at work and the trabeculae are clothed by a spindle-cell and osteoclast tissue; here both lacunar resorption and new bone formation are proceeding on an extensive scale, but if one is to judge from the results, the process of resorption finally gets the upper hand. In addition to this are the areas of osteoclast and spindle-cell tissue, often cystic. In most of these there are few, if any, calcified trabeculae left, but there is still an attempt at bone formation which results in the production of small irregular patches of coarse-fibred osteoid tissue.

With regard to the amount of calcium salts present in the apparently well-calcified trabeculae, it seems impossible to assess this by histological means. They certainly contained sufficient calcium to prevent the cutting of sections without some decalcification, but in most areas only a comparatively short decalcification in Müller's fluid was required. This easy decalcification may depend on the scantiness and fineness of the trabeculae rather than upon their calcium content.

2. **The Parathyroid Tumour.**—The naked-eye appearance of the specimen is well shown in *Fig. 475*, which also shows an apparently normal parathyroid removed at the same time. The cut surface of the tumour had a yellowish appearance and showed many small rounded and larger slit-like spaces. Many of these were vascular, some apparently cystic.

*Fig. 476* gives a good picture of the cellular structure of the tumour on the left hand and of the normal parathyroid on the right. The cells of the growth were arranged in a continuous compact mass only broken by numerous thin-walled vessels and some small cystic spaces, so that the



**FIG. 476.**—High-power view of a small area of the parathyroid tumour (left) and of a normal parathyroid (right).

tumour did not show the lobulated arrangement of the normal gland. Apart from this point, there is very little deviation from the normal structure, and the histological appearance is certainly compatible with a condition of hyperfunction.

From the point of view of its pathological anatomy this case presents several interesting features :—

*a.* Although classified as fibro cystic disease, the specimen shows no naked-eye evidence of any filling up of the marrow spaces by fibrous tissue, and cysts are found only in association with the tumour-like masses.

*b.* These tumour-like masses form a prominent feature of the specimen and might be mistaken for tumour deposits, but both their histological appearances and the other features of the case suffice to contradict this impression. There is little doubt that such masses merely represent an exaggeration and persistence of the active spindle-cell and osteoclast tissue which, as has been seen, covers the trabeculae in certain areas ; this tissue again appears to result from a hyperactivity of the processes which are normally involved in the destruction and remodelling of the bones.

c. The patchy nature of these areas of activity is a striking feature. The histological appearances in the neck of the femur (*see Fig. 472*) suggest that a zone of activity may travel through the bone, leaving in its wake an atrophied but quiescent bony structure. What factors determine these local activities? We know so little of the chemical and other factors which control bone formation and destruction that it appears impossible at the present time to offer any detailed explanation of the processes involved. Certain features of this case give a suggestion that minor injuries may play a part in determining the development of some of the areas of red tissue. Somewhat below the middle of the shaft of the tibia (*see Fig. 471*) two elongated tracks of pink tissue extend inwards at right angles to the cortex; these correspond to the site of insertion of the screws used in extending the limb. This injury has evidently determined the focal development of osteoclastic tissue.

### COMMENT.

It is most unfortunate that we have no record of the blood content immediately after the exarticulation of the left lower limb, but it might be reasonably expected that calcium mobilization would be temporarily much

TABLE SHOWING CALCIUM AND PHOSPHORUS BLOOD-COUNT OF L.W.\*

DATE	CALCIUM	PHOSPHORUS	ALKALI RESERVE	REMARKS
2.2.29	—	—	—	Fracture of left femur
3.2.29	15.9	3.0	—	
17.2.29	16.9	4.2	—	
4.7.29	—	—	—	Amputation of left lower limb
4.10.29	8.2	2.8	43.0	
8.1.30	11.6	3.3	—	
9.1.30	10.9	2.5	—	Removal of parathyroid tumour
10.1.30	10.5	2.8	43.0	Neuromuscular irritability
11.1.30	10.1	2.9	—	
12.1.30	9.9	2.7	—	Onset of definite tetany. (CaCl <sub>2</sub> .)
13.1.30	9.5	2.4	—	Tetany
14.1.30	9.4	3.1	—	"
15.1.30	9.1	3.7	57.0	"
25.1.30	9.6	3.9	—	Chvostek's sign positive
30.1.30	10.2	3.9	—	" " "
18.2.30	9.5	—	—	" " "
16.7.30	9.9	—	—	" " "
17.9.30	10.6	3.1	—	Chvostek's sign negative
9.9.31	11.0	2.9	—	" " "

\* A normal hospital or home diet was being taken.

reduced on removal of the major site of bone absorption and that consequently the serum level would be lowered. There is this curious discrepancy in the figures given in the table, that, whereas the calcium content of the blood was high in February, 1929, at the time when extensive changes were going on in the left femur following its fracture, nevertheless in October, 1929, when X rays demonstrated considerable progression of the disease, the blood calcium was only 8.2, and in January, 1930, when there was undoubted and rapid absorption, the blood calcium was only 11.6.

Administration of parathyroid hormone is followed by a complicated and as yet imperfectly understood series of physiological phenomena. Of these the more outstanding are a fall in serum phosphorus and rise in calcium, an increase in urinary output of calcium and phosphorus, and a decrease in the calcium content of the skeleton. The generally accepted theory of hyperparathyroidism suggests that when an excess of the hormone is in circulation calcium is mobilized from the skeleton, passes to the blood, where its normal level is raised, and is thence excreted from the system.

The second point which requires explanation is the onset of tetany when the serum calcium stood at 9.9 mgrm. per 100 c.e. Salvesen<sup>1</sup> states, "There is one characteristic feature of the tetany of parathyroid origin; the blood calcium is always below a critical level, which in dogs and human beings is about 7 mgrm. per 100 c.c. of plasma against the normal of 10 mgrm. All kinds of tetany with normal blood calcium can therefore be excluded as being caused by parathyroid insufficiency." Van Slyke appears to agree with this statement, which may be taken as representing the popular view.

Calcium is present in serum probably in three different fractions. In normal adult serum the total calcium content is given by Peters and Van Slyke<sup>2</sup> as 9.0–11.5 mgrm. per 100 c.e., and as estimated in our own hospital it is usually about 10.0 mgrm. Of this total about 4 mgrm. are in combination with protein and have no physiological function in our present condition; the rest is diffusible, and of this about 2.5 mgrm. are in ordinary solution as ionized calcium, the balance being held in solution by an unknown factor in some way related to the parathyroid hormone; this last quantity may or may not be ionized. The level of diffusible calcium also varies considerably with that of phosphorus. Consequently the significance of serum calcium estimations cannot be interpreted without knowledge of the phosphorus and protein levels. In this patient there is no reason to believe there was any gross renal damage since there was never more than a very faint trace of albumin in the urine. The phosphorus determinations were constantly rather low and would tend to cause reciprocally higher calcium values. It may be taken, therefore, that the total calcium values give in this case a fair indication of the diffusible or active calcium.

Had the calcium been greatly raised for a considerable period and then fallen rapidly to a much lower level, the tetany might be explained on a basis of altered tolerance. However, it fell only from 11.6 to 9.9 mgrm., so such an argument can here hardly be valid. In this connection Gold's case<sup>3</sup> affords an interesting comparison; removal of a parathyroid tumour was followed by a fall of serum calcium from 13.1 to 9.9 mgrm., and at no time was there any suggestion of tetany.

On the other hand Bulger<sup>4</sup> and his co-workers have reported a case similar to our own. Before removal of a parathyroid tumour the serum calcium stood at 16.4 mgrm.; on the fourth day after operation it was 11.3 and there were signs of neuromuscular irritability; on the seventh day it was 10.8 and there was obvious tetany. During the following fortnight the calcium varied between 4.1 and 5.5, and very large doses of parathormone and of calcium lactate by the mouth were given without any relief of symptoms, which became almost unbearable. On the twelfth day calcium

chloride was given intravenously and a gradual clinical improvement immediately started, so that in twenty-four hours the patient was almost symptom-free. The serum calcium, however, rose slowly, and a week later it was 7.1. Here, as in our own case, tetany bore little relation to the serum calcium level, and was relieved by calcium chloride and not by calcium lactate, although Bulger was able to show that much of the latter was absorbed.

Tetany may occur in a variety of conditions, which fall into two groups. In the one category, which includes hypoparathyroidism, osteomalacia, and infantile rickets, there is usually a low serum calcium. The other group comprises hyperventilation, loss of hydrochloric acid from vomiting, etc.; here the blood calcium is normal, but there is a measurable alkalæmia. In both types of case the tetany has a considerable clinical similarity, and yet to each is ascribed a separate pathogeny—thus the low calcium content on the one hand or the more alkaline blood on the other acts directly on nerve-fibres or their end-plates and increases their excitability. As a corollary of this it has been suggested that the injection of calcium chloride produces an acidæmia which neutralizes the existing action of a low calcium and inhibits tetany—surely rather a far-fetched argument.

Our own case and that of Bulger<sup>4</sup> show that total serum-calcium estimations, and probably also diffusible calcium estimations, may give but little information about the clinical condition of the patient, may be no guide to the rate of skeletal destruction, and may give no warning of the approach of tetany. It may be that an additional factor, at present undiscovered, must be introduced to account for the apparent dissimilarity in the etiology of these types of tetany.

Our thanks are due to the Directors of the Bland-Sutton Institute of Pathology and of the Courtauld Institute of Biochemistry, Middlesex Hospital, for their advice and criticism, and to Mr. Thornton Shiells and Mr. W. Pilgrim for the drawings which illustrate the paper.

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## THE INCIDENCE OF ANAEROBIC INFECTIONS IN THE GALL-BLADDER.

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IN a former paper<sup>1</sup> (1930) reporting a bacteriological study of the material from fifty consecutive cases of cholecystectomy, prominence was given to the frequency with which *B. welchii* can be cultivated from the biliary passages. Examination of a further fifty cases has confirmed this, and it is noticeable that when using a medium favourable to anaerobes other workers have cultivated *B. welchii* from a fair proportion of cases. Thus Williams and McLauchlan<sup>2</sup> (1930) obtained *B. welchii* from some part of the biliary passages in 6 out of 97 cases of acute and chronic cholecystitis. The assessment of the significance of anaerobic infection as a primary causal factor in lithiasis must be as speculative as is the assessment of the part played by other bacteria. But, as was emphasized in the previous series, and as has been stressed by Williams and McLauchlan, the overwhelming frequency of undoubted intestinal bacteria, of which *B. welchii* is one, definitely indicates that the intestine is the commonest source of gall-bladder infection, be that infection a primary or a secondary lithogenic factor. This view is opposed to that of Rosenow<sup>3</sup> (1916) and of Wilkie<sup>4</sup> (1928).

There can be no doubt that modern investigators are much more thorough in their technique than were the earlier workers. The latter concentrated their attention mainly on the fluid contents of the gall-bladder and thus missed a large number of infections. It is, therefore, useless to compare the statistical studies of recent times with those of the past century. The publication of this second series of fifty cases may emphasize the necessity for routine anaerobic cultures in any investigation of gall-bladder infection.

**Technique.**—Fresh material only has been used. Gall-bladders which have been removed more than two hours before receipt in the laboratory have been regarded as unsuitable. The gall-bladder has been received in the laboratory wrapped in sterile gauze.

Primary cultivations have been made in glucose-broth, meat-broth, litmus-milk, and on ordinary agar, as follows:—

**Bile.**—The external surface of the gall-bladder was seared and the fluid contents removed by puncture with a Pasteur pipette.

**Gall-bladder Wall.**—After dissecting off any fat and peritoneal covering (if practicable) a large piece of the underlying tissue, including the mucous membrane, was excised, washed in saline, and implanted.

**Stones.**—These were washed in running water for twenty-four hours,



soaked in 5 per cent phenol for half an hour, transferred to alcohol for a few minutes, and then flamed. The whole stone was then incubated in meat-broth for forty-eight hours, and if growth occurred the sterilizing process was repeated. If sterile, the stones were crushed and implanted. In a few cases with soft stones it was found impossible to sterilize the external surface, but it was presumed that the process of washing had removed any contaminating bile.

**Type of Material.**—Ten specimens were from acute cases with a frankly congested, hæmorrhagic, and sometimes gangrenous gall-bladder. The bacteriological findings in these cases are starred in the table. *B. welchii* was present in five of these (three in pure culture). Two cases which had the so-called ‘Persian carpet appearance’ (see the illustration, Gould and Whitby, 1927<sup>5</sup>) contained, not *B. welchii*, but *B. coli* in pure culture.

BACTERIA ISOLATED FROM GALL-BLADDER WALL, FROM BILE,  
AND FROM STONES.

CASES.			ANALYSIS.
All three infected	..	7	2 enterococcus; 1 <i>B. welchii</i> *; 1 <i>Sta. albus</i> ; 3 mixed ( <i>B. coli</i> * and <i>B. welchii</i> , <i>Str. viridans</i> and <i>B. lactis aerogenes</i> , <i>B. coli</i> * and enterococcus and <i>B. welchii</i> ).
Wall only	..	20	7 <i>B. coli</i> (4*); 2 <i>B. welchii</i> *; 2 enterococcus; 1 <i>Sta. albus</i> ; 8 mixed (2 <i>B. coli</i> and <i>B. welchii</i> , 2 enterococcus and <i>B. welchii</i> , 3 enterococcus and <i>B. coli</i> , 1 <i>B. coli</i> * and <i>Str. viridans</i> ).
Wall and bile only	..	7	1 enterococcus; 3 <i>Sta. aureus</i> ; 1 <i>B. paratyphosus B</i> ; 2 mixed (2 <i>B. coli</i> and enterococcus).
Stones only	..	3	1 <i>B. welchii</i> ; 1 <i>Sta. aureus</i> ; 1 mixed ( <i>B. coli</i> and <i>Sta. albus</i> ).
All three sterile	..	13	
		50	

\* Acute cases.

Incidence of various organisms in 50 cases, including mixed infections: *B. welchii* 10; *B. coli* 18; enterococcus 13; *Sta. aureus* 4; *Sta. albus* 3; *Str. viridans* 2; *B. paratyphosus B* 1; *B. lactis aerogenes* 1.

No cases of strawberry gall-bladder are included. Many specimens showed evidence of recent inflammation, and two, both infected, were of mucocele of the gall-bladder. Of the 13 sterile cases, 11 were small thickened gall-bladders with a few stones, but 2 were of the type which a naked-eye examination would have suggested as being probably infected.

### COMMENTS.

The incidence of *B. welchii* in this series is 10 out of 50, and in the first series was 9 out of the same number. These figures are considerably higher than those of other workers. There is no doubt that a deep tube of litmus-milk is the most sensitive medium for detecting this organism. Undoubted intestinal bacteria were present in 31 cases. Of the balance of 6 positive cases, 4 were *Sta. aureus* and 2 *Sta. albus*. Both these organisms may be found in faeces from time to time. In the two cases where *Str. viridans* was found it was associated with intestinal bacteria. One case of *B. paratyphosus B*

was found in this series, and one in the previous fifty cases. Thus it is clearly evident that intestinal bacteria are those most frequently found in gall-bladder infections.

The constancy of intramural infection as compared with other parts of the biliary system is agreed by all modern workers, but it is doubtful whether experimental work on animals has as yet proved either the true pathological process in cholecystitis or the exact route by which infection, primary or secondary, finds its way to the gall-bladder. It is necessarily difficult to reproduce human conditions in animals, which have different habits, diet, and posture, as well as relatively uncomplicated lives.

One of us (L. E. H. W.) is indebted to the Medical Research Council for a grant for the performance of this work.

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**FURTHER STUDIES IN INTRAVENOUS PYELOGRAPHY.**

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THE object of these studies was to investigate further the advantages and limitations of intravenous pyelography with uroselectan. One of us (R. J. W.<sup>21</sup>) has already recorded his earliest experiences. We feel that the selected cases described below may be deemed of sufficient interest to be worthy of record in view of the facts that intravenous pyelography is still in its infancy and that only by the accumulation and consideration of recorded experience can the true value of this new method of diagnosis be properly assessed.

**HISTORY.**

It may be of interest to survey briefly the history of the attempts that have been made to delineate the outline of the interior of the urinary tract with media opaque to the X rays, at first by the injection of such substances through the ureter catheter, and more recently by virtue of their excretion by the kidneys following intravenous or oral administration.

Voelker and Lichtenberg<sup>16</sup> in 1906 were the first to report on the use of such a substance, introducing colloidal silver into the renal pelvis through the ureter catheter, thus giving to urology the science and art of pyelography. They found that this substance, in addition to being expensive, gave rise to unpleasant, and at times alarming, constitutional reactions. But despite its use for some years by one of us (R. J. W.) no untoward effects have ever been experienced.

Kelly and Lewis<sup>7</sup> in 1913 were the first to suggest the use of a halogen compound, and recommended silver iodide emulsion, claiming that, in addition to being less irritating and safer than the colloidal silver, it was also much cleaner to work with.

Braasch and Mann,<sup>2</sup> working at the Mayo Clinic in 1916, demonstrated that many silver compounds, when retained in the pelvis of the kidney or injected under pressure, produced areas of cortical necrosis. Other reports by Praetorius<sup>12</sup> (1919), Schüssler<sup>14</sup> (1920), and Barreau<sup>1</sup> (1921) have confirmed the opinion of Braasch and Mann in pointing out the danger and unsuitability of injecting silver compounds into the renal pelvis.

Burns,<sup>3</sup> of the Johns Hopkins Hospital, in 1915 reported satisfactory results with thorium nitrate, the aqueous solution being neutralized by sodium hydroxide in the presence of a sufficient amount of sodium citrate to prevent the precipitation of the thorium. Later<sup>4</sup> he made an extensive report on his further investigations regarding the use of this medium, which gained

considerable popularity until Cameron<sup>5</sup> in 1918 published his paper on pyelography with the iodides of potassium and sodium as his media. He pointed out that the thorium nitrate solution was expensive, had to be prepared with care, and that times arose when it was difficult to obtain.

Weld,<sup>18</sup> in the same year, suggested the use of sodium bromide, claiming that this solution possessed all the properties of the ideal pyelographic medium, i.e., it lacks toxicity, is non-irritating, easily obtainable and sterilized, keeps well under all conditions, and is reasonable in cost. The introduction of these two substances, sodium iodide and sodium bromide, formed an important step in the development of pyelography, as shown by the fact that they have been used almost exclusively by urologists since their introduction. While working on the toxicity of pyelographic media, Weld<sup>19</sup> found that intravenous administration of as much as 50 c.c. of a 25 per cent solution of sodium bromide to a dog weighing 6 kilo. produced no toxic effects, the experiment proving the remarkable lack of toxicity of this substance. At the same time he reported a death following the use of thorium nitrate, the substance introduced by Burns.

Osborne, Sutherland, Scholl, and Rowntree,<sup>11</sup> in 1923, took advantage of two facts, the first that sodium iodide is normally excreted by the kidneys, the second that at that particular time this substance was being given intravenously in large doses to patients at the Mayo Clinic for the study of the therapeutics and pharmacology of the iodides. Where they found no idiosyncrasy to these compounds they injected intravenously 5 to 20 gm. of a 10 per cent solution of sodium iodide; and afterwards took X-rays of the urinary tract. They found that the bladder showed up satisfactorily in nearly every case, and got fair X-ray shadows of the kidneys and ureters in half the cases. They carried their investigations a step further, and found that after oral administration of the halogen compound they obtained satisfactory radiograms in half their cases. This work, then, marked the beginning of pyelography following the intravenous administration of a substance opaque to the X rays and the introduction of an important new chapter on the history of the diagnosis of disease of the urinary tract.

There began to appear in the literature the results of the efforts of workers to perfect this easy method of obtaining an outline of the urinary tract, a method needing only the knowledge of venepuncture and obviating the necessity of the cystoscope, an instrument whose use will always remain in the hands of a select few.

Volkman<sup>17</sup> in 1924 tried the effects of many preparations, but with unsatisfactory results.

The next worker to publish results showing a distinct advance on this new method of pyelography was Roseno,<sup>13</sup> who used sodium iodide and urea as his medium, giving it the name of 'pyelognost'. After experimenting on dogs with this new substance he gave it to patients, and obtained pyelograms in eighty cases. The disadvantage of this medium lies in the large dosage necessary, rendering a constitutional reaction likely in a certain number of cases.

Ziegler and Köhler,<sup>22</sup> in 1930, tried to obviate the effects of intravenous administration of Roseno's medium by giving it orally, but they obtained

their pyelograms only after compression of the ureters with an abdominal pad for some time, a factor which might well lead to misrepresentation of the true state of the interior of the renal pelvis and calices.

The most recent advances on this aspect of urological diagnosis are associated with the names of Swick,<sup>15</sup> von Lichtenberg,<sup>10</sup> Binz, and R  th. Binz, a pupil of the celebrated Ehrlich, working with R  th in Berlin on organic compounds of iodine, produced a substance belonging to the pyridine group and gave it the name of 'selectan-neutral'. This was originally intended to show on the X-ray film the outline of the gall-bladder after the medium had been administered intravenously, but that viscus failed to show, and instead a shadow of the renal pelvis and ureter appeared, of sufficient clarity to justify and inspire further work along these lines. Selectan-neutral itself was not very satisfactory as it was somewhat uncertain, and mild reactions sometimes followed its use. Eventually 'uroselectan' was produced from selectan-neutral by substituting sodium-glycin in place of a methyl group. It was first introduced by von Lichtenberg and Swick at the General Meeting of the German Society of Urology held at Munich in 1929. We see that its appearance was the result of the patient investigations of many workers extending over a number of years, and not the sudden inspiration of genius.

The first cases in this country were reported by Kidd<sup>8</sup> in March, 1930, to be followed soon afterwards by those of Heritage and Ward.<sup>6</sup>

Since then another—and simpler—substance has appeared on the market, namely 'abrodil', made by Bayer. It has the advantage of containing a higher percentage of organically combined iodine than uroselectan, so that a smaller quantity can be used; it is also more soluble in water. Both are innocuous, although Whorlow<sup>20</sup> says that he has seen a case of ulceration of the arm following the intravenous injection of uroselectan, apparently due to extravasation of the substance into the tissues around the vein. In our experience we have never seen a single untoward reaction, either general or local, follow the injection of uroselectan or abrodil.

### CASE REPORTS.

We have now used uroselectan in the investigation of many cases with symptoms referable to the urinary tract, and from these we have selected six as being of particular interest. In the descriptions of them appended below we have used the terms 'ureter catheter pyelography' and 'intravenous pyelography', both of which phrases are more convenient and less verbose than 'pyelography by injection of opaque medium through the ureter catheter' and 'pyelography following the intravenous administration of uroselectan'.

For the intravenous pyelography in this series of cases we used only uroselectan, manufactured by the firm of Schering-Kalbaum, of Berlin, while for the ureter catheter pyelograms sodium iodide (13½ per cent) was the medium injected.

*Case 1.*—E. G., male, age 67. Admitted on Feb. 17, 1931.

**HISTORY.**—During the past three years intermittent attacks of pain beginning in the right posterior renal area and radiating round the loin into the iliac fossa. H  maturia was noticed first six months previously, and continued intermittently

up to the time of admission; sometimes associated with the pain mentioned above, at other times painless. He had occasional nocturnal frequency during the previous three years. There were no symptoms of uræmia.

ON EXAMINATION.—General condition fair, right kidney tender, enlarged, nodular, and moving with respiration. Urine: microscopic blood.

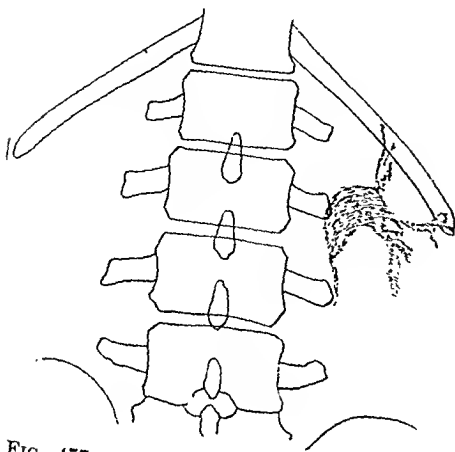


FIG. 477.—Case 1. Uroselectan pyelogram.

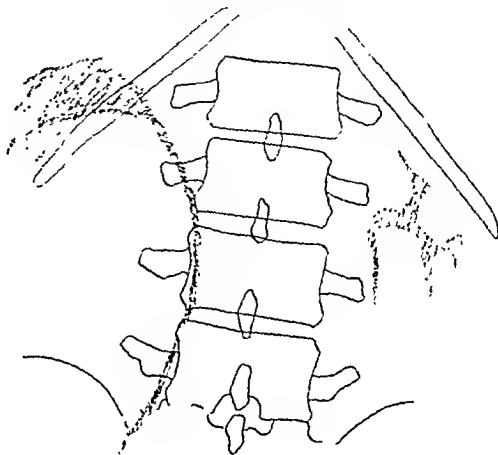


FIG. 478.—Case 1. Ureter catheter pyelogram.

**X-ray of Urinary Tract.**—Nothing abnormal noted.

**Cystoscopy.**—Showed ulceration around the internal meatus; the ureteric orifices were normal, but they could not be catheterized on this occasion.

**Uroselectan Pyelogram (Fig. 477) showed:—**

**Left side.**—Pelvis enlarged, middle and lower calices normal and well filled. Upper calix gives a fainter shadow but does not suggest a filling defect.

**Right side.**—No shadow.

**Ureter Catheter Pyelogram (Fig. 478) showed:—**

**Left side.**—Normal.

**Right side.**—The lower and middle calices are represented by indefinite shadows. The upper calix is distorted and displaced downwards. The upper third of the ureter is displaced inwards, while the pelvis is displaced upwards.

**DIAGNOSIS.**—Neoplasm of the lower pole of the right kidney.

**OPERATION (Feb. 23).**—Enlarged right kidney removed. Shown on section in Fig. 479.

**PATHOLOGICAL DIAGNOSIS.**—Hypernephroma of lower pole of the kidney.

**Comment.**—Taking the clinical evidence of this case and the uroselectan pyelogram alone, the conclusion was fairly obvious that we were dealing with a pathological right kidney, but beyond that it was impossible to go. A pyelogram following cystoscopy was necessary to make the diagnosis definite.



FIG. 479.—Case 1. Portion of removed kidney.

**Case 2.**—S. W., age 50. Admitted on March 3, 1931.

**HISTORY.**—Urinary symptoms for twelve months previously. Pain in intermittent attacks beginning in the right posterior renal area, radiating round the loin into the iliac fossa, but of no great severity. Nocturnal frequency to a moderate

extent, one to three times. She had noticed for a long time that her urine contained a deposit on standing. No hæmaturia.

**ON EXAMINATION.**—General condition good, right kidney palpable on inspiration, not definitely enlarged; no tenderness or fixity. Urine contained microscopic pus.

**X-ray of Urinary Tract.**—Nothing abnormal noted.

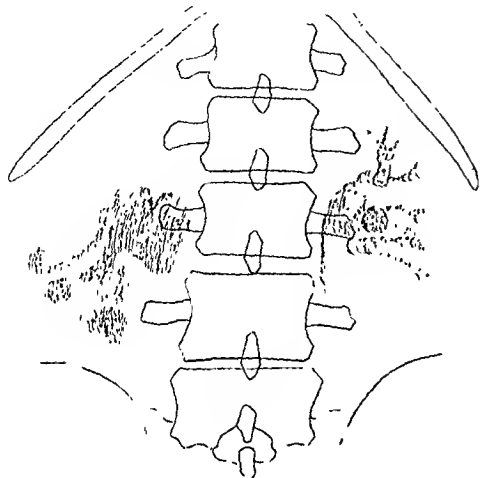


FIG. 480.—Case 2. Ureter catheter pyelogram.

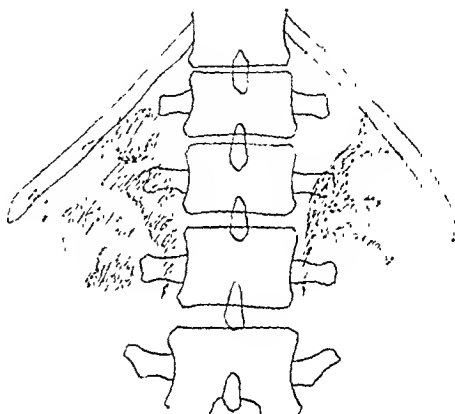


FIG. 481.—Case 2. Uroselectan pyelogram.

**Cystoscopy.**—Sacculation marked behind right ureteric orifice and also on the left wall of the bladder. Both ureteric orifices were normal in appearance; both sides were catheterized.

**Ureter Catheter Pyelogram (Fig. 480) showed:—**

**Left side.**—Calices elongated but not pathological.

**Right side.**—Pelvis dilated; lower calices have filled, but the movement of the patient gives them a blurred outline. The middle and upper calices not filled.

**Uroselectan Pyelogram (Fig. 481) showed:—**

**Left side.**—Normal.

**Right side.**—Marked dilatation of all calices and pelvis.

**DIAGNOSIS.**—Hydronephrosis.

**OPERATION (March 15).**—Right kidney exposed. Hydronephrotic; aberrant vessel passing across the lower part of the renal pelvis into the lower pole and constricting the ureter. Nephrectomy performed. Specimen shown on section in Fig. 482.

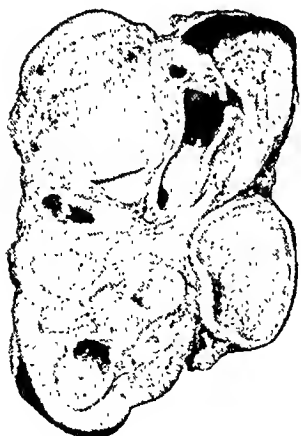


FIG. 482.—Case 2. Portion of removed kidney.

the possibility of neoplasm into one's mind. All the dilated calices showed up so beautifully in the uroselectan pyelogram that the diagnosis then became obvious.

**Comment.**—This case provides an interesting contrast to the one above, for on the use of uroselectan depended the final and correct diagnosis, whereas in Case 1 ureter catheter pyelography was found necessary for an accurate diagnosis. Although hydronephrosis could be suspected from the pyelogram following cystoscopy, yet the absence of the middle and upper calices brought





of the renal parenchyma, found its exit into the ureter blocked by the stone, and this no doubt accounts for the good density of the shadows. It is so well known that an obstruction to the flow of the uroselectan down the ureter often leads to a shadow of increased density in the calices and pelvis that the ureters are sometimes compressed as they pass over the pelvic brim through the medium of a pneumatic bag placed over the lower abdomen and distended under a firm band.

Interest lies in the fact that both the ureter catheter pyelogram and that obtained from the uroselectan failed to indicate the presence of congenital cystic disease. It is true that on the left side the calices are elongated, but the cupping is normal and the appearance on this side seems to be more indicative of compensatory hypertrophy of this kidney, which one would expect in view of the marked hydronephrotic condition of the other.

Although the extensively damaged kidney apparently required removal, this could not be entertained, as the view was taken that congenital cystic disease is always bilateral.

*Case 4.*—T. M., male, age 41. Admitted on April 10, 1931.

**HISTORY.**—Intermittent attacks of left-sided renal colic for past four years. Last attack in December, 1930; since then periodic discomfort in the left kidney region. The relation of his colic attacks to exercise was indefinite, but rest would nearly always relieve the pain. No frequency of micturition or passage of blood, stone, or gravel.

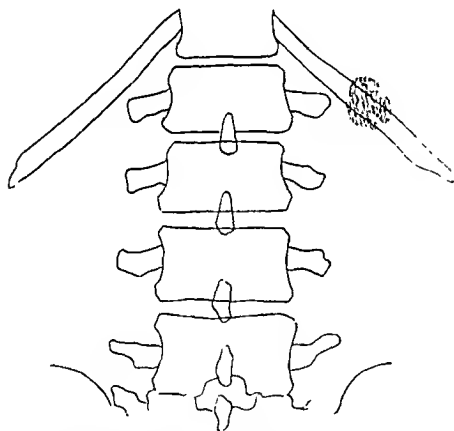


FIG. 486.—*Case 4.* Rounded shadow shown by X rays. ? Calculus.

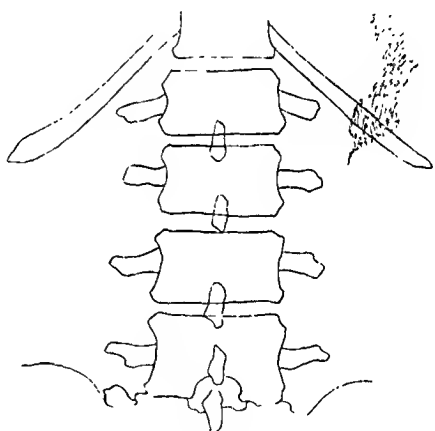


FIG. 487.—*Case 4.* Uroselectan pyelogram.

**ON EXAMINATION.**—Healthy-looking man. Gross dental sepsis; abdomen showed slight tenderness on deep pressure over the line of the left ureter.

**X-ray of Urinary Tract.**—Showed a rounded shadow, about an inch in diameter and of even density, in region of left renal pelvis, probably a calculus (*Fig. 486*).

**Uroselectan Pyelogram** (*Fig. 487*) showed:—

**Right side.**—Shadow of fair density showing normal outline of pelvis and calices, not shown in diagram.

**Left side.**—Only upper calix filled and shows dilatation; shadow of calculus in position of, and apparently filling, the pelvis.

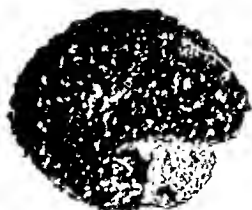


FIG. 488.—*Case 1.* Uric acid calculus removed.

# INTRAVENOUS PYELOGRAPHY

629

*Ureter Catheter Pyelography* was not done.

**OPERATION** (April 16).—Left kidney exposed and pelvis incised from posterior aspect. Uric acid calculus removed (Fig. 488). The upper and middle calices were dilated.

**Comment.**—This case is really included in the series because of the contrast it makes with the following one.

**Case 5.**—G. C. A., male, age 36. Admitted on April 10, 1931.

**HISTORY.**—Numerous intermittent attacks of left-sided renal colic during past six months, becoming more frequent. Last attack two days before admission. As

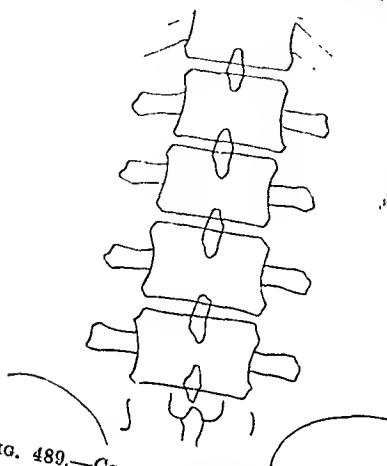


FIG. 489.—Case 5. Triangular shadow shown by X rays.

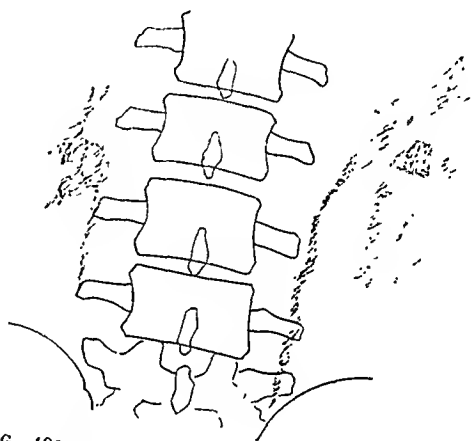


FIG. 490.—Case 5. Uroselectan pyelogram.

in the last case, the patient was indefinite as to the relation of the attacks to exercise and rest. There were no associated urinary symptoms such as frequency of micturition or hæmaturia.

**ON EXAMINATION.**—General condition good. Carious lower teeth; slight tenderness on pressure in left posterior renal area.

**X-ray of Urinary Tract** (Fig. 489).—Showed triangular shadow, not of great, though of even, density in the left kidney area, slightly lateral to the usual position of the pelvis. It was thought to be a calculus in the middle calix.

**Uroselectan Pyelogram** (Fig. 490) showed:—

**Right side.**—Normal shadow.

**Left side.**—Ureter well shown. In kidney area, around shadow of suspected calculus, are several indefinite shadows.

**Ureter Catheter Pyelography** was not done.

**OPERATION** (April 16).—Left kidney exposed. Lower pole slightly enlarged and surface studded with

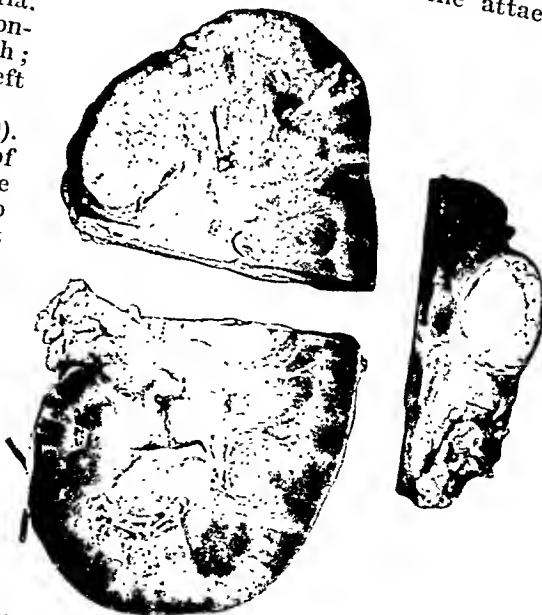


FIG. 491.—Case 5. Kidney removed at operation

miliary tubercles. The 'calculus' proved to be an area of hard caseated and encapsulated tuberculous material. Nephrectomy performed. The specimen is shown in Fig. 491.

**Comment.**—This case is of interest for the following reasons. Apart from the fact that the patient had no history of tuberculosis and was in excellent health, there was no painless frequency, that common harbinger of renal tuberculosis, nor had he passed blood, either macroscopic or microscopic; further, no pyuria was present. In view of the 'closed' type of his tuberculosis shown by the encapsulated caseated mass, the above findings are not remarkable, but there was nothing 'closed' about the numerous miliary tubercles in the lower pole of the kidney. The specimen shows them extending throughout the renal tissue from capsule to calix.

We feel that a ureter catheter pyelogram would probably have cleared up the diagnosis. As it was, no harm was done, because exploration of the kidney, necessary for removal of a stone, was equally necessary for the tuberculous condition found.

Perhaps we should have assumed that the ill-defined shadows over the area of the affected kidney meant a damaged renal function, but here our experience agrees with that of others, that in those cases where, because of renal incompetence there is little or no shadow in the kidney area, there is usually no sign of the ureter either. In this case, however, it was quite well defined.

*Case 6.*—C. M., male, age 49. Admitted on April 19, 1931.

**HISTORY.**—For several months the patient had been troubled with a dull aching pain in the left loin, coming on usually after he had started work, and relieved by rest. There was hæmaturia on two occasions, the blood being mixed with the urine; the first occasion was six months, the second three days, before admission. There was no frequency of, or pain on, micturition: the stream was of good calibre and the general health good.

**ON EXAMINATION.**—General condition good. There was slight tenderness over the left kidney, which was not palpable. Urine: Deposit of urates.

**X-ray of Urinary Tract (Fig. 492).**—Showed a huge kidney-shaped shadow of great and even density in the left kidney area. It extends from the upper border of the last thoracic vertebra to the lower border of the third lumbar, and its breadth is about half its length.

**Cystoscopy.**—Nothing abnormal seen in bladder. Both sides catheterized.

**Ureter Catheter Pyelogram (Fig. 493)** showed:—

**Right side.**—Some dilatation of pelvis. Calices and ureter normal.

**Left side.**—Normal outline of ureter seen extending downwards and inwards from a point about an inch medial to the lower pole of the shadow seen on the X-ray film of the urinary tract.

**DIAGNOSIS.**—Large calculus of left kidney distending and filling up the pelvis.

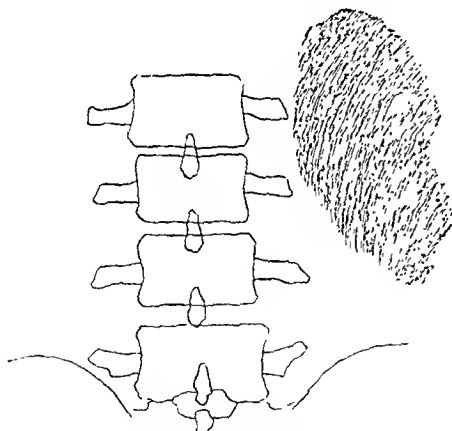


FIG. 492.—Case 6. Large shadow shown by X rays.

*Uroselectan Pyelogram (Fig. 494) showed:—*

*Right side.*—Similar to ureter catheter pyelogram, but the shadow is not so dense.

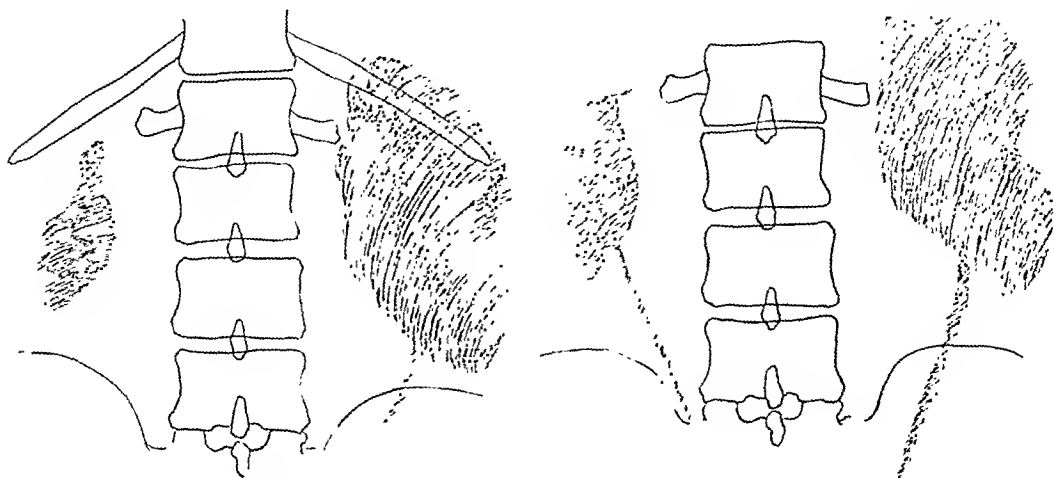


FIG. 493.—Case 6. Ureter catheter pyelogram.

FIG. 494.—Case 6. Uroselectan pyelogram.

*Left side.*—The film has been taken at a higher level and shows a small shadow, due to the medium, capping the upper pole of the shadow of the calculus.



FIG. 495.—Case 6. Kidney removed at operation.

**OPERATION.**—Left kidney exposed; pelvis greatly distended and filled with hard globular mass. Nephrectomy. Specimen shown in Fig. 495.

The stone, composed of uric acid, is the largest we have ever seen in a kidney. It measures 4 in.  $\times$  3 in.  $\times$  2 $\frac{1}{4}$  in., and weighs 15 oz. The pelvis is enormously distended and much thickened. The kidney tissue, except for the upper pole, has been mostly destroyed.

**Comment.**—In this case the ureter catheter pyelogram made the diagnosis clear. The intravenous pyelogram was not only successful in doing likewise, but also gave us the additional information that some functioning kidney tissue was still present (a fact confirmed by examination of the extirpated kidney), for uroselectan must have been excreted in order to appear in the ureter. The point raised, however, is more of academic than of practical interest, as it is obvious from the size of the stone that nephrectomy is the only form of treatment for such a huge renal calculus.

### CONCLUSIONS.

We have as yet had no untoward reactions, either general or local, resulting from the use of uroselectan.

At the present time intravenous pyelography is often disappointing owing largely to its uncertain results. It sometimes fails to show a shadow even when other investigations show the kidneys to be healthy. Even when successful the shadows are often of insufficient density to show the finer detail of the calices, making diagnosis exceedingly difficult or impossible even to those skilled in the reading of pyelograms.

In some cases, particularly when there is obstruction to the outflow of urine down the ureter, it is likely that intravenous pyelography will often give more information than ureter catheter pyelography, as in Cases 2 and 3 of this series. Such cases, however, constitute only a small proportion of the total in the average urological clinic.

We feel that the future of intravenous pyelography is one of distinct promise. Already von Liechtenberg<sup>9</sup> has indicated that other substances have been prepared of which less quantity has to be given, yet which are more certain to produce the shadows indicating to us the normal or the pathological, whichever is the case. Just as cholecystography has been simplified by the oral administration of the phthalein-halogen compounds, so there is no reason to doubt that in due course, following the indications already given by the work of Osborne and others<sup>11</sup> and of Ziegler and Köhler,<sup>22</sup> clear pyelograms will be obtainable without difficulty after the oral administration of radiologically opaque substances.

Our thanks are due to Dr. Whately Davidson, Physician-in-Charge of the X-ray Department, Royal Victoria Infirmary, Newcastle-upon-Tyne, for the radiograms published in this article.

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633

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## THE SYMPATHETIC IN ACUTE GENERAL PERITONITIS. A CLINICAL STUDY WITH OBSERVATIONS ON TREATMENT.

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To balance correctly the possibilities in the human mechanism between distorted function resulting in a pathological state and a pathological state resulting in distorted function is often extremely difficult. To the clinician, however, it is of supreme importance, since in the first case by correcting function the lesion is cured, and in the second case, removal or cure of the lesion corrects function. This difficulty presented itself to me in the understanding and treatment of acute general peritonitis, and the investigation which resulted has furnished findings which seem to be of considerable interest. Although as yet I am by no means content with the perfection of the interpretation of these findings the clinical application of them has been encouraging.

It is not considered necessary for the purpose of this article to enumerate the many lesions which may result in a general peritoneal infection, but I wish to emphasize that the subject under consideration does not include such abdominal crises as accompany primary obstruction of the lumen of the bowel. In these latter where shock or toxæmia may predominate, treatment, both operative and therapeutic, must vary to attain success.

**Clinical Picture.**—Familiar as is the picture of a subject of general peritonitis to the eye of every clinician, it is felt that this paper would be incomplete were a description of it omitted. More particularly is this the case since a correlation of the clinical and operative data led me to formulate the hypothesis upon which I finally based treatment.

The case described is that of a man admitted to hospital with a duodenal ulcer perforated twenty-four hours. A short quick operation is performed, and for some hours afterwards the general and abdominal distress considerably abates. The pulse-rate falls several points. Pain is not now complained of. Following the first outpouring of seropurulent fluid, this settles away and only a drop or two damps the gauze adjacent to the extruding drain. The true peritonitic picture next appears. The pulse increases in rate and gets steadily thinner. It loses its thrust. The face becomes grey, and the cheeks shrink in against the underlying bones. The brow is puckered, the eyelids even in sleep barely close, and death-like strips of white sclera remain visible. Breathing is short and fast. The skin is cold and wet, but the mouth is dry and the tongue furred. The teeth are visible between lips too dry and shrunken to close. Occasionally a black vomit is gulped up. The abdomen is tightly distended, and everything is still within it. A day or two passes, and a frightened look appears in his eyes. He becomes anxiously restless, and

a feeling of great warmth causes him to push down the bedclothes. The skin remains quite cold. Neither fæces nor flatus are voided and no urine is passed. Without assistance, from two to four days from the onset of acute symptoms, he dies. In the last few unconscious moments the grip of the syndrome that has crushed the patient to death often relaxes, and what all efforts have failed to do now happens: fæces and flatus are voided incontinently, but it is too late.

*Explanations of this Picture.*—A review of the literature bearing on this subject makes it clear that there is a lack of agreed opinion as to the progress of events which culminate in this condition. It is generally agreed, however, that in an established case of general peritonitis an acute intestinal obstruction exists. As to the nature of this obstruction, a mist of doubt and uncertainty falls around the endeavours at explanation. In the past decade it would appear that the views expressed by Wilkie have engaged the most attention, and only within recent times have research workers and clinicians allowed their thoughts to focus in other directions. In 1921 Wilkie stressed the idea that the obstruction was most often organic in type and resulted from plastic adhesions, generally in the pelvis. This opinion has led to the devising of many mechanical operative procedures to relieve the obstructed bowel. The more recent departures from these views have engaged less the attention of research workers and clinicians, in particular the latter. Such information as we find calls for clinical observation and investigation of the possible part taken by the sympathetic system in the production of the peritonitic syndrome. It will therefore be my endeavour to show briefly how investigation led me beyond given facts to the adoption of the views expressed by Hurst and such workers as Alvarez and Hosoi.

**Observations on the Organic Theory.**—In the post-mortem room it can be demonstrated that plastic adhesions form between coils of peritonitically obstructed bowel and that these are often most formidable in the lower reaches of the abdomen. If they are undone in the living subject and recovery follows it would seem certain that they were the cause of the obstruction. Further, if the small intestine is opened, either to drain it or to feed the patient, or both, and recovery result, it would suggest a mechanical difficulty in the passage of contents along the bowel. A closer scrutiny of various happenings relative to this obstruction tends, however, to shake one's confidence in its organic nature. In the first place mechanical relief of the obstruction alone by no means invariably led to a cure. Further, I had noted that under certain conditions the obstruction appeared to be relieved before any mechanical interference had been attempted. This was seen when a spinal anæsthetic was given, and again just at death. On one or two occasions, also, when administering an intravenous saline to a patient apparently completely obstructed, abdominal rumblings were heard, and later an incontinent motion was passed. Such happenings could not be explained by a mechanical obstruction, and demanded further investigation.

**Direct Observations of the Peritonitic Abdomen.**—The interior of the abdomen presents the picture of a lymphatic space submitted to an invasion by bacteria or chemicals, or both. Little enlargement on this finding need be made unless to stress the feature of tissue immobilization. The diaphragm



shortens its course, and the liver, kidneys, and spleen are at rest. Even the bladder may cease to function.

Attention is next focused on the obstructed intestine. Conditions of working and anxiety for the patient as a rule made accurate observation difficult, but in the main findings were constant. In an early peritonitic obstruction the distension is greatest in the small intestine, the large bowel being little if at all involved. The duodenal and jejunal regions are more distended than the lower ileal coils, whilst the last few inches of ileum appear least affected, or may be in spasm. Even where the lower ileal spasm was not very apparent it was repeatedly found that only with great difficulty could the contents of the distended areas be made to pass towards the ileocolic region. There seemed little doubt that in an early peritonitic abdomen the obstruction was not organic. Any lymph deposits present lay between coils of intestine, were soft, and easily swept away by a finger. The points where intestinal contents were held up were at the pylorus, the duodenojejunal flexure, and the ileocecal region. In late cases conditions were found unchanged except that the colon had become involved, though always, apparently, to a less extent than the small intestine. The situation of obstruction in it was found to be at the left brim of the pelvis or the pelvirectal area.

These direct observations substantiated many of the recognized procedures in dealing with such a case: that the stomach cannot empty and must be emptied reversely; that feeding must be done by a jejunostomy; and that an enterotomy gives quicker and more certain relief to the obstruction than a cæecostomy; that an enterocolostomy is preferable to a cæecostomy; and that the latter if used must be accompanied by the passage of a rubber tube several inches through the ileocecal valve. Finally, it confirmed what to most has become a conviction—that all one can expect to accomplish by means of enemas is the exhaustion of the patient. Their passage beyond the pelvirectal area is obstructed.

**Physiology.**—When the findings described are scrutinized it is seen that there is not one area of obstruction but several, and each of them is associated with a junctional sphincteric area in the intestinal tract. At each point of obstruction a hypertonic action in the musculature of the sphincter appears to be present, with at the same time an inert paralytic and distended condition in other parts of the bowel. Such a selective antithesis of events is not explained by a mechanical or organic obstruction where the contents of the bowel pack up against the obstruction. Disturbed function alone supplies a scientific interpretation, and this, aided by the work of recent investigators, I shall endeavour to give.

The motility and functioning of the intestinal tract is automatic and intrinsic, response being made both to intake and quality of food. In the past it has been held that this automaticity of action depended on a perfect balance between the motor and inhibitory nerves. Canon and Hosoi, however, have now demonstrated the falseness of this latter theory, and have shown that changes in the motor function of the intestine result from sympathetic activity only. If this finding is applied to an experiment carried out by Alvarez and Mahoney, a further deduction is possible. They

injected chemical irritants into the ileocecal region, thus producing patches of peritonitis, and found that the bowel for some distance above the cæcum was empty and very irritable and the upper jejunum and duodenum became distended with fluid. I also find that Elliot by stimulation of the splanchnic nerve produced contraction of the ileocolic sphincter. It may then be deduced that peritoneal irritation resulting in a sympathetic excitation can produce the complex picture both of spasm and inhibition or dilatation in the bowel. Further, it may be noted that junctional sphincteric hyperactivity and sequelæ similar to that found by Alvarez and Mahoney in laboratory investigation can also be seen clinically in cases of chronic appendicitis leading to the so-called appendix dyspepsia. I have noted the same syndrome with tuberculous mesenteric glands as the underlying cause. It is a well recognized fact that if this distorted function of the bowel in cases of chronic appendicitis, and still more in cases of subacute appendicitis, is forcibly thwarted by purging, the inflammatory condition of the appendix is quickly aggravated. This aptly demonstrates the association between the offending lesion and the functional departure from normal in the bowel. The complex effect of sympathetic excitation appears to be towards immobilization of bowel activity, and its purpose is protective of lesions without the lumen of the bowel.

The collection of these facts now made it possible to explain my findings in the general peritonitis as being the result of a sympathetic crisis. The degree of sympathetic excitation from a general peritoneal irritation had produced a functional obstruction in the bowel. In the kidney the vessels upon which kidney function depends had become contracted and so the quantity of urine secreted diminished. The vessels of the skin became contracted and the sweat glands hyperactivated. Thence is seen the pallid, wet, salty skin of the peritonitis and consequently the tissue dehydration and the excessive excretion of chlorides from the body.

**Treatment.**—The formulation of this conception as to the nature of the intestinal obstruction in acute general peritonitis provoked a review of the methods of treatment. It may well be argued that danger would await any departure from the tried mechanical methods of its relief. This I felt in my work, and only slowly as confidence increased did I depart from their use.

It would appear clear that if a successful treatment of acute general peritonitis is to be evolved there are three elements in the condition towards which attention must be directed: (1) The infected peritoneum—the source of sympathetic excitation; (2) The hyperactivated sympathetic system—the protective syndrome; (3) The resulting depletion of body chlorides and fluids and the concentration of urea and other toxins in the blood.

The infected peritoneum calls for observation from two directions, an infected lymphatic space and the source by inflammatory irritation of sympathetic excitation. Successful treatment must master the infecting elements, yet in no way increase the peritoneal irritability. Operative interference is usually imperative the moment such a case is detected. This must be planned so as to reduce irritation of the peritoneal surfaces to the minimum. Such procedures as require handling of loops of bowel or pawing around the inflamed abdominal cavity should, wherever possible, be avoided. Swabs and packs should never touch the peritoneum. Suffice it to say regarding

the operative methods adopted, after a careful investigation of the case, where a small incision and the insertion of a soft rubber drain meets the situation, that is the treatment of choice. There is little doubt that in my series of cases the adoption of this severe conservatism has given me a patient in a much better condition for post-operative therapy.

The intraperitoneal abscess is treated as are abscesses in other parts of the body. Warm glycerin fomentations spread across the entire abdomen increase the local congestion and are extremely effective in the relief of pain. Where anaerobic organisms are either known or suspected to have invaded the peritoneum the injection into the cavity of sterile glycerin (5 to 10 c.c. on two consecutive days) in my experience helps the resolution of the inflammation. Systemic treatment to combat the infection has been variously tried, and, difficult though it is to estimate benefit, it would seem that intramuscular or intravenous anti-gas-gangrene serum alone gives good results. This I have adopted as a routine therapy in cases of general peritonitis. Its benefit points to the frequent inefficiency of the autogenic defences against anaerobic infections.

I now pass to the treatment of the hyperactivity of the sympathetic nervous system. Where its exciting agent, the bacterial or chemical irritation of the peritoneum, can be quickly dealt with little attention is required. Unfortunately not every case of general peritoneal infection does quickly yield to the direct attack on the infecting agents, and the entire syndrome of sympathetic agitation demands attention. Though it is agreed that an acute intestinal obstruction is then present, I have found it wise to be cautious. The obstruction is present as part of the physiological immobilization and is evidence that the peritoneal infection is not in hand. The first duty, therefore, is not to undo this immobilization but rather to abet it, making at the same time other conditions as favourable and safe for the patient as possible. To do this, the stomach and rectum—the only areas of the bowel approachable—should be emptied prior to operation. At the same time,  $\frac{1}{8}$  gr. of morphia and  $\frac{1}{100}$  gr. of hyoseine are given hypodermically. Following the operation the  $\frac{1}{8}$  gr. of morphia is continued four-hourly. Should any consequent sign of vomiting appear, it must be instantly relieved by stomach lavage. These methods maintain the patient in comfort and his strength is conserved.

Here I would digress to deprecate methods of treatment of this stage, that have become established—though perhaps falsely—by use. First there is the giving of fluids by mouth or rectum for the abatement of thirst. It is doubtful if any absorption can take place from the stomach, and fluids, whether given by the mouth or rectum, in the early stages of a peritonitic obstruction merely aggravate vomiting. The mouth should be cleansed and should be washed out when desired, but nothing swallowed. Again, anything suggesting an enema, whether nutrient or for purposes of attempted emptying of the bowel, must be avoided. The result of such well-meant interferences appears to be to set up a reverse peristalsis, a form of intestinal movement all too easy to arouse in a nerve-obstructed bowel. The one essential in the early phases of the obstructed peritonitis is to obtain a satisfactory hypnosis: it conserves strength, it abates thirst, and aids the physiological processes at work.

The most difficult problem I have met in the course of treatment is how far to persist with this disregard for the obstructed bowel combined with the progressive dehydration of the patient. Though I am now satisfied that too early interference is both futile and detrimental to progress, accurate indications that a change in tactics should be adopted are extremely difficult to demonstrate. That there is a correct moment seems certain, since I have observed that treatments vary in effect in accordance with when they are applied. Too early replacement of the fluid loss is futile, as it is very quickly thrown out, not merely by the skin but by a renewal of vomiting. Also it is possible only very temporarily to raise the chloride content of the blood. A selective sympathetic depressant would overcome these difficulties, but as yet none is known. The moment to be determined is undoubtedly that at which the progress of the infection has been checked and the danger from it, both local to the peritoneum and general, has passed. That moment correctly gauged, together with the nullification of the activity of the abdominal sympathetics, will rescue the patient back to life. I have noted that any sign of macroscopic pus, in a wound or from a drain, is a certain indication that treatment may proceed; and in seven cases where the patient was *in extremis* and the subjective sensation of excessive warmth had developed, I stepped in with happy results. There still remain cases, however, where a surer indication of this stage is yet wanted, and at present I depend to some extent on a gradually acquired clinical knowledge of it. It might be argued that the stage in treatment I am seeking to determine is that at which the course of the disease, without further aid, would normally turn to betterment. This, however, I have seen to be erroneous. It is the stage where one was wont to consider further operative interference essential. Instead, I now direct my efforts against the sympathetic excitation.

Not only the sympathetic action in the bowel but its general effects also must be annulled. Isolation of the bowel from stimuli and restoration of its function does not alone render the patient in all cases beyond danger. The departures from normal in the blood and the arrest of renal function constitute an equal danger. Hence the first stage of treatment must be pushed to extremes so that the rescue may not be nullified by continued and irrepressible sympathetic activity. When we proceed to isolate the bowel from sympathetic stimuli it may be accomplished by the use of atropine. Opium in all forms must be stopped and its place taken by atropine sulphate,  $\frac{1}{100}$  gr. every four hours. The stomach is then washed out, and in order to stimulate forward peristalsis raw meat juice is introduced. This latter is continued by swallowing. One to two pints of normal saline with 4 per cent gum acacia are now given intravenously. All other routes of administering saline have proved unsatisfactory. Hypertonic solutions are recommended by many, but I now give normal saline for the following reason. It is necessary that a multiplicity of sympathetic reflex arcs should be depressed, and as the action of atropine is not directly on the sympathetic system I introduce the bromides as a further aid. Their absorption and action are greatly increased where some degree of chloride deficiency remains in the blood.

Provided the management of the treatment to this point has been well timed, it not infrequently happens that an almost or actually incontinent

motion of the bowel takes place. At all events, in from one-half to one hour the picture of the peritonitis begins to change: the tissues begin to relax and fill out; the immobile parched tongue shows signs of moisture and movement; and an ear to the still abdomen will detect noises. Two hours following the change in treatment the first enema is given, slowly and by gravity. It must be stopped if there is any sign of upper abdominal pain or vomiting. If some dehydration is still taking place, a second saline, normal or hypertonic, should be repeated ten hours after the first. The first natural bowel motion is often involuntary. It would appear from an analysis of subjective sensations that the early peristalsis takes the form of long contractions beginning in the small intestine and passing in a continuous wave towards the rectum. When this stage is reached, anxiety as to the patient's life may cease. One condition, however, is worthy of mention. Where correction of the effects of the sympathetic activity produces an indifferent result on the patient's condition and doubt remains as to the quiescence of the peritoneal infection, a blood transfusion should be given. Observation suggests that the best time for this is to replace the whole or part of the second saline transfusion by blood. Not till then may its necessity become apparent.

The final stage in the treatment is to bring under control the enterocolitis which results from excessive growth of bacteria within the bowel during the phase of obstruction. Generally this is easily accomplished. The return of normal secretions and bowel function are the main requisites. Charcoal and bismuth aid in soothing the ulcerated mucosa.

### CASE REPORTS.

I shall now describe briefly some of the conditions to which I have applied this mode of treatment. My total number of cases where the entire syndrome of sympathetic excitation had to be dealt with is not large, and apparently is becoming less. I can account for the diminishing numbers in two ways only: the conservative operative measures I now adopt in all cases of general or potential general peritonitis; and the institution, immediately after, or even before operation, of the measures I have described to assist the physiological processes at work to combat the peritoneal irritants. Not infrequently it is found that by these methods an apparently established case of general peritonitis subsides rapidly about the third day after operation without the institution of any treatment to suppress the sympathetic activity.

*Case 1.*—L. A., male, age 48. Admitted to hospital on Jan. 18, 1929. Temperature 97.4°, pulse 140. Face a blue and grey colour. Vomiting a black vomit: abdomen tightly distended. History that he began five days previously with pain low down in right side of abdomen. Had taken castor oil. Was brought directly to hospital by his relatives without calling doctor. Became unconscious in transit. Perforated appendix with general peritonitis diagnosed.

Morphia,  $\frac{1}{8}$  gr., given and continued four-hourly. Surrounded with hot bottles. Stomach washed out. Enema given but no result.

4.30 p.m. (five hours after admission). General condition improved: temperature 100°, pulse 146.

5.30 p.m. Open ether anaesthetic. Right paramedian incision and drain inserted. Bowel seen to be red and angry and bathed in pus.

2nd day.—10 a.m. Temperature 101°, pulse 132. Mouth very dry, regularly cleansed, but nothing swallowed. Abdomen tightly distended. Dressing changed.

# ACUTE GENERAL PERITONITIS

641

Glycerin soak applied across abdomen; 8000 units anti-gas-gangrene serum given intramuscularly. Morphia continued.

3rd day.—10 a.m. Looked extremely ill. Temperature  $101.4^{\circ}$ , pulse 146. Respirations short and quick. Face grey and drawn. Skin clammy. Abdomen tightly distended. Voice feeble. Passing no urine. Vomited at 4 a.m. and 9.30 a.m. Stomach washed out; 8000 units serum given intramuscularly. No discharge from drain.

4th day.—10 a.m. Restless and mentally confused. Pushing down bedclothes and complaining of warmth. Temperature  $99.4^{\circ}$ , pulse thin and running. Morphia stopped. Atropine,  $\frac{1}{100}$  gr., given and continued four-hourly. One pint normal saline with gum acacia given intravenously. Sodium bromide, 15 gr., by mouth with meat-juice. One hour later a glycerin soap and water enema gave a coloured result. 3 p.m. Tissue dehydration less. Tongue slightly moist. Mentally clear and not restless. Slight rumbling audible in abdomen. Some discharge from abdominal drain.

6 p.m. One pint normal saline with gum acacia intravenous transfusion. One hour later an enema produced a result. Veronal given at 11 p.m. and 4 a.m. 5th day.—10 a.m. Somewhat restless night. Passed 4 oz. of urine at 6 a.m. and 6 a.m. only. Some tissue dehydration continuing. True pus seen at edge of drain. One pint of normal saline with gum acacia given intravenously. Bowel moved incontinently half an hour later. Atropine stopped. Recovery after this was uninterrupted, and the patient left hospital healed on the thirty-fifth day. On the fourteenth day an appendix concretion escaped from the wound.

Case 2.—G. C., male, age 50. Admitted to hospital on Jan. 18, 1930, eighteen hours after an abdominal injury at sea. When first seen the temperature was  $97.6^{\circ}$  and the pulse 112. Abdomen tightly distended. The blow, he thought, was on the left side of the upper abdomen and was immediately followed by great pain. A general peritonitis following traumatic rupture of stomach or bowel was diagnosed. As he was very collapsed it was decided to delay operation. Morphia,  $\frac{1}{2}$  gr., was given and hot bottles were applied round the patient. Three hours later his condition had improved.

As the chest condition was doubtful it was decided to give  $\frac{1}{100}$  gr. of atropine along with  $\frac{1}{2}$  gr. of morphia prior to anaesthesia. Warmed ether by Shipway's apparatus was given. Through a short left paramedian incision the abdomen was opened. Gas escaped. A seropurulent exudate lay amongst tense and inflamed coils of bowel. No perforation was found on the anterior surface of the stomach. Some blood was seen on displacing the transverse colon upwards. A tear in the transverse mesocolon was exposed, but the exact degree of stomach rupture was not ascertained. The condition of the patient became critical. A small drain was inserted down to the opening and the abdomen quickly closed. He was returned to bed and heat applied. Two hours later  $\frac{1}{2}$  gr. of morphia was repeated, and was continued every four hours.

2nd day.—10 a.m. General condition improved. No vomiting. Temperature  $102^{\circ}$ , pulse 118. On changing the dressing it was found that much serum had escaped from the drain. Abdomen tightly distended; 10,000 units anti-gas-gangrene serum given intravenously. Glycerin fomentation to the abdomen. I thought that, though at an earlier stage than usual, an intravenous saline might continue the general improvement. This, however, had to be stopped after introducing 2 oz. as the patient complained of upper abdominal pain and threatened to vomit. A burst of perspiration broke out shortly afterwards which left the patient weaker than before.

6 p.m. The full peritonitic picture now established. When sleeping his eyelids scarcely met. His cheeks were sunken, and dry teeth could be seen between contracted lips. The abdomen was tightly distended and immobile. 3rd day.—10 a.m. Temperature  $101.2^{\circ}$ , pulse 138. Dressing changed. Only a small quantity of serum had escaped from the drain. Morphia continued. Mouth kept clean and washed out. Nothing swallowed.

4th day.—10 a.m. Condition now critical. Restless and pushing down bed-clothes. A look of fear in his eyes. Had vomited a mouthful of smelling black vomit at 8 a.m. No urine passed for twelve hours. Morphia stopped and  $\frac{1}{100}$  gr. of atropine four-hourly commenced. Stomach washed out and meat-juice and 10 gr. of sodium bromide given. One pint of normal saline with gum acacia administered intravenously. Towards the end of the saline a prolonged rumbling was heard in the abdomen and the patient thought his bowel had moved incontinently. This, however, did not happen. Soon after the saline he experienced a short attack of shivering with an increase of perspiration. Two hours later with a glycerin soap and water enema a coloured result was obtained and some flatus passed.

6 p.m. Condition improved. Temperature  $100.4^{\circ}$ , pulse 110. Still losing some fluid. No sickness. Had passed a little urine. Meat-juice and bromide continued by mouth. An intravenous normal saline again given, and an enema later produced a fair result.

5th day.—10 a.m. Much improved. Tissues now filling out and tongue moist. Abdomen distended, but rumblings heard. Purulent matter at edge of drain. Atropine stopped.

6 p.m. During afternoon a small unformed motion had been passed by the bowel with some flatus.

From this time there was nothing unusual about the recovery, and the patient left hospital healed on Feb. 21.

Case 3.—Mrs. M., age 36. One month married. Admitted to hospital on Dec. 10, 1930. History that two days following a normal menstruation she was seized with severe pain in the rectum which later was also felt in the right iliac region. Looked very pale and ill. Temperature  $100^{\circ}$ , pulse 124. Per vaginam there was excessive tenderness of the cervix, but nothing unusual was otherwise found. There was no discharge or bleeding. The lower half of the abdomen was distended without rigidity or tenderness on palpation. I decided to observe the case. At first she improved, but on the sixth morning there was a quick relapse. Pulse and temperature rose. The rectal pain, which had never quite disappeared, became severe. Per vaginam the cervix was extremely tender, and for the first time a boggy feeling was present behind the posterior fornix. The right fornix felt somewhat full though not tender on bimanual examination. The abdomen had become generally distended though not rigid. I decided to explore.

Warm ether anaesthetic. Through a mid-line incision the lower abdomen was opened. A fetid brown serum lay amongst distended coils of bowel. Turning the latter upwards it was seen that the right ovary and broad ligament were gangrenous and that gangrene was spreading into the left broad ligament. The peritoneum of the pouch of Douglas was black, and on opening it a quantity of foul-smelling serum and clot was evacuated. The left ovary, both tubes, and uterus appeared unaffected. The diagnosis appeared to be invasion of a hæmatoma in the rectovaginal space by anaerobes, with a spreading gangrene in the adjacent cellular tissues. As the patient was almost pulseless, operative procedures had to be limited and rapid. The right infundibulo-pelvic ligament and ovarian ligament were tied off, and the intervening tissue, including the ovary, was removed. The gangrenous contents of the left broad ligament were cleared out. A drain was inserted into the rectovaginal space and the abdomen closed. The patient was returned to bed, heat applied, and  $\frac{1}{6}$  gr. of morphia four-hourly commenced.

2nd day.—10 a.m. Temperature  $100^{\circ}$ , pulse 132. Abdomen distended, but no vomiting. Foul-smelling gangrenous discharge from drain; 10,000 units anti-gas-gangrene serum given intramuscularly. Frequent mouth-washes given.

10 p.m. 10,000 units of serum repeated.

3rd day.—10 a.m. Temperature  $100.6^{\circ}$ , pulse 134. Mouth dry but kept clean. Nothing swallowed; 10,000 units serum given. A glucose saline per rectum was tried, but produced vomiting.

4th day.—10 a.m. Temperature  $101^{\circ}$ , pulse 140. Very little from drain. Abdomen tightly distended and causing much discomfort. Ten e.c. of sterile glycerin injected into pelvis through the drain. Glycerin fomentation applied across

the abdomen. Much relief of discomfort followed; 5000 units serum given. Unable to pass urine. Catheterization produced 6 oz.

6 p.m. 5000 units serum repeated.

5th day.—10 a.m. Vomited at 4 a.m. and 9 a.m. Constantly complaining of burning in epigastrium. Stomach tube passed. Much comfort resulted. Discharge from drain more than previous day and less foetid. Five c.c. of glycerin introduced into pelvis. Fomentations continued; 5000 units serum given.

6th day.—10 a.m. Looks extremely ill. Temperature 99.8°, pulse thin and running. In spite of continued morphia complains much of rectal pain. Picture of dehydration now marked. At dressing discharge greatly increased and occasional flakes of macroscopic pus seen in it. Morphia stopped. Atropine,  $\frac{1}{100}$  gr., given and continued four-hourly. One pint of normal saline and gum acacia given intravenously together with 10,000 units of anti-gas-gangrene serum. A slight shiver and sweat followed the saline, but there were no signs of sickness. Bromide, 10 gr., and meat-juice given by mouth. A little later slight rumblings in the abdomen could be felt by the patient. Two hours afterwards an enema gave a small watery result and a little flatus was passed.

7th day.—10 a.m. Temperature 100.8°, pulse 118. Still somewhat dehydrated, but taking meat-juice and glucose freely by mouth. At dressing the abdomen seemed much less tense. Discharge from drain now free. Odour almost gone and contains plentiful flakes of pus. Atropine and fomentations stopped, sodium bromide, 10 gr., with mag. carb. pond., 15 gr., continued four-hourly by mouth. An enema during the day produced no vomiting and gave a loose and satisfactory result. Flatus passed.

From this point the patient made a rather stormy but satisfactory recovery. A blood transfusion was required on Jan. 19, 1931, to correct her anæmia, and a retained abscess in the lower right iliac region required opening. She left hospital with a small suprapubic sinus on March 5.

By the method here described I have treated general peritonitis in 39 cases. These include: general peritonitis from ruptured appendix (19 cases), pneumococcal peritonitis (5), ruptured subphrenic abscess (1), puerperal peritonitis (2), traumatic rupture of the stomach (1), perforated duodenal ulcer (10), and gangrenous pelvic cellulitis (1). Amongst these, one case of pneumococcal peritonitis and the two cases of puerperal peritonitis required blood transfusion. In all the complete syndrome of acute general peritonitis was present. There was one death, from bronchopneumonia, of a duodenal ulcer patient, perforated twenty-six hours.

**Drugs.**—A few remarks may be made concerning the drugs I have used and those I have avoided. In the first stage morphia is the only medicament. It has been pointed out that dosage in excess of  $\frac{1}{2}$  gr. each four hours should be avoided. This is because it has been noted that greater quantities, though often apparently indicated, instead of lessening, tend to aggravate vomiting. This action of morphia, given in large doses in the peritonitic subject, has also been substantiated by Alvarez and Hosoi. In recent cases I have used barbitone derivatives to assist the morphia with encouraging results.

In the second stage of treatment, though atropine has proved unreliable in the hands of some workers, it has proved effective when employed at the stage in treatment here suggested. Atropine has no action on the normal tonus of the sphincters, but on the other hand hypertonus is abolished. The fitting explanation of this occurrence is that atropine has no direct action on the nerve-supply to the intestine. Its action is on the receptor centres in the bowel, and these it anaesthetizes from stimuli. Sympathetic stimuli



remain but are intercepted, and the intestine is thrown back on its intrinsic motility.

The chief difficulty in the use of the bromides is their method of administration, which must be by mouth. They are, however, very rapidly absorbed, and in the presence of a chloride deficiency doses as small as 5 gr. appear to give results.

One drug notable by its absence in this article is pituitrin. Because of its physiological action on the intestinal musculature, its use in this form of obstruction remains prevalent and is recommended. If, however, it is agreed that the junctional sphincteric hypertonus is the chief obstacle to the passage of contents, its value must be reconsidered. Doubtless it might produce some activity in the relaxed musculature, but being non-selective in its action it can only increase the sphincteric hypertonus.

The withholding of fluids in the early stages of treatment calls for mention. It is true that the dehydration produces discomfort to the patient and a certain degree of danger to life. If, however, it is agreed at this stage that fluid can neither be retained nor used to benefit, and merely aggravates that most exhausting symptom vomiting, it seems wise to withhold it. Water swallowed or ice sucked makes the thirst worse. Sedatives alone seem to appease it adequately.

In conclusion, I should like to mention that there is a type of peritonitis, which is purely streptococcal, where this mode of treatment would not apply. In it, though a very dangerous peritonitis is present, the symptomatology and reaction to the infection are quite different from the average general peritonitis. A flushed face and a clean moist tongue are present with a raised pulse and temperature. At first a little discomfort is complained of in the abdomen, with a little sickness, and a vague tenderness may be elicited on deep palpation. The bowel continues to move, and in the late stages a soft tumidity of the abdomen is apparent. Sympathetic excitation in such a case is absent or numbed from the onset. In a recent case of this condition where I opened the abdomen there was little or no bowel distension or inflammation, though a sticky lymph, flaked with pus, lay on its surfaces. The nature of this streptococcus is being investigated.

Further, I would emphasize that this treatment evolved is, in its application, clinical and not mathematical. The indication for adopting each step must first be present.

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## A CASE OF RAPIDLY-GROWING CARCINOMA IN THE NECK, ARISING IN A PARATHYROID REST.

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SINCE the discovery of the parathyroid glands by Sandström<sup>1</sup> in 1880, a number of tumours, both innocent and malignant, have been described as having their origin in these glands.

Ewing<sup>2</sup> describes both intra- and extrathyroid tumours arising from parathyroid tissue, including simple adenomata and malignant types, and he points out that in those tumours arising within the substance of the thyroid it is difficult to establish a parathyroid origin unless the tumour presents very definite structural characteristics. He also refers to the histological similarity of parathyroid tumours to the hypernephromata.

Gye<sup>3</sup> reports a case of primary malignant tumour of a parathyroid in a woman of 29, with recurrent metastases eleven months after its removal by excision. In a very comprehensive review of the literature of this subject he analyses the position and sites of origin of all primary malignant tumours of the parathyroids reported up to 1929. These are few in number, and in many of the recorded cases both their origin from parathyroid tissue and their malignancy are in doubt. Moreover, in all these cases the tumour arose in the region of the thyroid gland, with which normally the parathyroids are in close contact, or in whose substance they are actually embedded.

The tumour described in this paper is believed to have arisen in parathyroid tissue, and, from its site of origin, to furnish evidence of the occurrence of aberrant tissue of this nature above the level of the greater cornua of the hyoid bone. Supernumerary parathyroids in relation to the thyroid gland are not uncommon,<sup>4</sup> and the presence of isolated 'rests' has been inferred from the occurrence of parathyroid tumours deeply situated behind the trachea or even in the mediastinum,<sup>5</sup> in situations other than in association with the thyroid gland. Developmentally the occasional persistence of such aberrant tissue might reasonably be expected to be found at any point along the course of descent of the parathyroids.

The embryological development of the parathyroid glands is generally agreed upon. The upper one arises in the fourth visceral cleft close to the thymic bud which arises in the same cleft, but which later as a rule disappears. The lower one arises with the thymus in the third visceral cleft and descends on the dorsal aspect of the thymic pouch from the region behind the future posterior pillar of the fauces to its adult position behind the lower pole of the thyroid gland (Keith<sup>6</sup>). According to Cunningham,<sup>7</sup> "as a result of its caudal

migration, the upper part of the thymus becomes drawn out and finally disappears. It is in this process that the lower parathyroid is involved, as it is attached to the upper end of the migrating thymic rudiment—the part which disappears. The relative time of this disappearance determines the permanent level of the lower parathyroid, for until this happens the gland is dragged in the wake of the thymus.”

This explains the exceptional presence of the lower parathyroid at levels so variable as the bifurcation of the common carotid artery (where it is apt to be confused with the carotid gland) and the upper part of the thorax. In adult life the thymic-lower-parathyroid track is represented by a line parallel to and just in front of the anterior border of the sternomastoid muscle. It is more than conceivable, therefore, that fragments of parathyroid tissue could be found as ‘rests’, or that a complete parathyroid gland could be found arrested along the course of its descent. From such a ‘rest’, or from such an arrested lower parathyroid gland, situated above the level of the greater cornua of the hyoid bone, we believe the tumour described below to have arisen.

### CASE REPORT.

**HISTORY.**—J. J., age 49, a railway clerk, eight years ago noticed a small lump about the size of a grape, on the left side of the neck. It was a round, movable, painless mass, not tender to touch. It remained stationary, giving rise to no inconvenience until July, 1930, when it began rapidly to increase in size.

His previous history was uneventful. He played the violin until he was 25 years of age, and noticed repeatedly what he described as a ‘small knot’ in his neck at the point where his violin was held. Its site was identical with that of the lump which appeared later.

**ON ADMISSION.**—The patient was admitted to the Glasgow Royal Cancer Hospital in October, 1930. On admission his general condition was good. He was a sturdily built man with a rather florid complexion. There was slight widening of the palpebral fissure, but no obvious exophthalmos, tremor, or increased pulse-rate. Examination of the cardiovascular, respiratory, and urinary systems revealed no abnormality. He had always been a non-smoker and a teetotaler. The Wassermann reaction was negative.

**ON EXAMINATION.**—On the left side of the neck, just behind the angle of the mandible, and projecting from under cover of the upper end of the sternomastoid muscle, there was a visible swelling, about the size of a large hen’s egg, and of the same shape, with its apex directed upwards. It extended downwards and inwards into the anterior triangle of the neck to the level of the upper border of the thyroid cartilage. It was hard in consistency, and fixed both to the sternomastoid muscle and to the deeper structures of the neck. The neck was otherwise normal; the thyroid gland was not obviously enlarged. Endoscopic examination of the mouth, nasopharynx, and upper end of the œsophagus revealed no gross pathological condition.

**OPERATION** (Oct. 21, 1930).—Under general anaesthesia a transverse incision was made in the neck over the most prominent portion of the swelling. This revealed what appeared to be a tumour about the size of a hen’s egg, whitish in colour, hard in consistency, lobulated on the surface, encapsulated by a thin membrane, and firmly adherent to the surrounding structures. The major portion of the tumour was excised, together with part of the sternomastoid muscle, which was obviously infiltrated by the growth, but the deeper part (which was infiltrating the deep tissues of the neck and firmly fixed to the internal carotid artery) had to be left *in situ*.

When the tumour was exposed a segment was excised for rapid histological

examination by means of frozen sections. It was then noticed that the central portion of the tumour was soft and necrotic, and it was thought that bacterial infection with pus formation might have occurred. The pathological report on the frozen sections and on smears of the necrotic material indicated that a malignant tumour was present, but of an unusual histological type, and that there was no evidence of suppuration.

Radium needles of 1 and 1.5 mgrm., with active length of 2 cm. and 3 cm. respectively, were inserted round the cavity. In all, twelve needles, screened with gold, containing 15 mgrm. of radium were given, the dosage being 1600 mgrm.-hours. The wound healed without trouble, and the patient left hospital twenty-four days after operation.

**SUBSEQUENT PROGRESS.**—In December, 1930, he reappeared with a mass in the same position, but larger than before and extending downwards as far as the level



FIG. 496.—Lateral view of the tumour as it appeared in March, 1931.



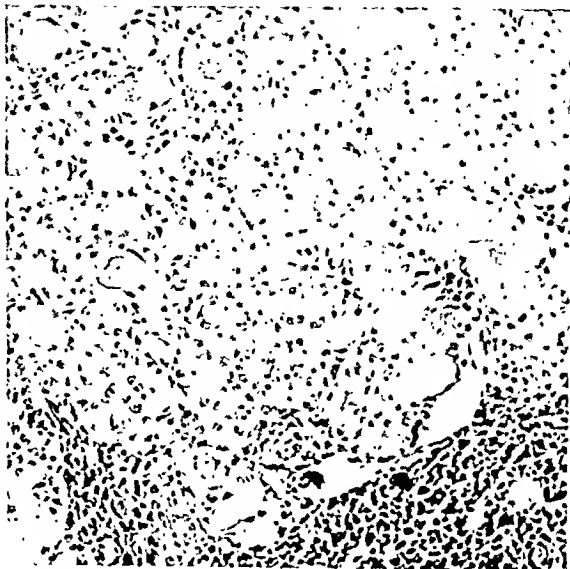
FIG. 497.—Lateral view of the tumour as it appeared in May, 1931.

of the second ring of the trachea. Radium needles of the graduated type (0.5 mgrm. per cm. of active length) were introduced into and around the growth, and were left *in situ* for 150 hours. In all, thirty needles containing 40 mgrm. of radium were used. There was no response, and by February, 1931, the mass had considerably increased in size and occupied a large area on the side of the neck. Moreover it had now commenced to fungate through the overlying skin.

Deep X-ray treatment was adopted, but without improvement in the local condition, and on March 31, 1931, the tumour showed signs of local metastasis (Fig. 496). On May 2, 1931, an intravenous injection of 20 c.c. of soluble sodium fluorescin was given half an hour before the administration of deep X-ray therapy. This treatment was repeated at intervals of a week, four treatments being given in all. During this time the tumour increased rapidly and progressively in size, and by the end of May, 1931, the left side of the neck was occupied by a large mass of tumour tissue which involved the skin overlying an area extending almost to the mid-line anteriorly, and to the posterior border of the sternomastoid muscle

posteriorly, and limited above by the angle of the mandible, and below by the clavicle. Over this area the skin presented a tense, rather shiny surface, and was raised up into a number of rounded excrescences varying in size from that of a walnut to that of a small pea (*Fig. 497*). This appeared to be due to the coalescence of numerous discrete nodules of growth. In colour the skin in this area was bluish or pinkish, and in several places it had become softened and necrotic. From the resulting fissures a thin greenish-coloured pus freely oozed, and could be expressed in quantity on slight pressure. The skin surrounding this nodular suppurating area was deeply pigmented owing to applications of deep X-ray therapy. Apart from the main mass of tumour in this region, there were a number of small discrete nodules lying at a distance of from half to one inch from the sharply circumscribed edge of the tumour.

In the anterior triangle, on the right side of the neck, there were similar nodules, some of which, however, were detectable



*Fig. 498.*—Section showing the lobulated structure of the tumour, with acini containing colloid. (Low power.)

only by palpation. Further extension of the growth was apparent over the manubrium and upper part of the sternum, where a number of papules could be seen. Over the xiphisternum there was a single larger nodule which was freely movable over the underlying bone.

The right side of the chest was the site of a very extensive metastatic deposit which had infiltrated from within the thorax, and presented a nodular mass which displaced the nipple upwards and outwards, and, at one point, showed signs of fluctuation. Smaller masses were present in the right axilla.

The patient left hospital, at his own request, on June 6, 1931, and died shortly afterwards. A post-mortem examination could not be obtained.

**HISTOLOGY.**—The earlier sections, taken at operation on Oct. 21, 1930, showed two types of growth. A single block, out of several prepared on this occasion,



*Fig. 499.*—Note the palisade arrangement of cells, the deeply stained nucleoli, and the amorphous material resembling colloid.

yielded sections which differed in histological features from those from the remaining

blocks. These latter sections were in all respects similar to those obtained on subsequent occasions.

The single block referred to showed a tumour composed of cells resembling those of normal parathyroid. These were arranged in palisades, each being four or five cells deep. Between the palisades there was a very fine fibrous reticulum. The tumour was subdivided by coarser bands of fibrous tissue into large irregular circumscribed nodules, giving the appearance of solid acini. Here and there the tumour had an alveolar structure, the centre of the alveoli being occupied by an amorphous material resembling colloid. These alveoli were relatively scanty, and the 'colloid' was small in amount (*Fig. 498*).

The cytoplasm was relatively abundant, having a finely granular or reticulated structure, resembling the pale-staining 'watery cells' of the normal gland. Mitotic figures were fairly abundant. The nuclei were large, oval in shape, rich in chromatin, and each having a distinct deeply-staining nucleolus. Occasional larger cells were seen with oxyphil granules in the cytoplasm (*Fig. 499*).

The greater part of the tumour which was originally removed, and all portions which were subsequently removed for biopsy, presented a somewhat different histological picture. The cells had a similar arrangement, but showed marked vacuolation of the cytoplasm, and there was a complete absence of any colloid-like material.

To enable the minute structure of the tumour to be studied in detail, a sufficiently large portion was removed at operation on April 1, 1931, so as to allow a number of different fixatives to be employed. Frozen sections were also prepared, both from fresh and from fixed material. The histology showed a tumour composed of vacuolated cells. There was a scanty stroma of fibrous tissue, mostly devoid of nuclei, with relatively few young fibroblasts, and containing some delicate blood-vessels. The tumour cells were large, closely packed, polyhedral in shape, and arranged in palisade form. The columns forming larger or smaller lobules radiated from a central necrotic zone. The lobules were in many cases several deep.

In sections stained by the routine method this central zone was seen to be composed of necrotic pink-staining cellular debris with a few pyknotic nuclear remains. Frozen sections, stained with haematoxylin and Scharlach R showed that the central zone was composed of cells that had undergone fatty degeneration (*Fig. 500*). The cells in the immediate neighbourhood of the central zone, for an extent of four or five layers, also exhibited fatty degeneration. This was also well seen in sections prepared from portions of the tumour fixed in the vapour of osmic acid. Attempts to demonstrate the presence of glycogen in the tumour cells were without success.

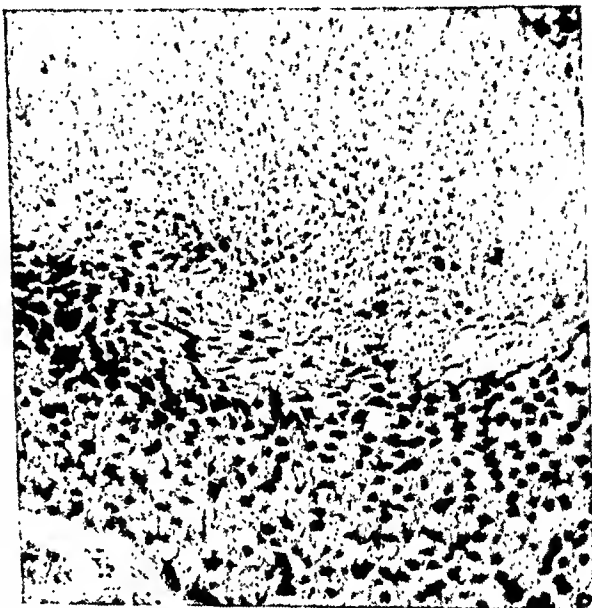


FIG. 500.—Frozen section showing central zone of fatty degeneration. (High power.)

## DISCUSSION.

The case recorded in this paper is one of tumour formation in the neck, arising in a situation where, normally, no parathyroid tissue occurs, but

which, from its histology, we believe to be of parathyroid origin. Clinically, the course of the disease proved rapidly fatal, as the rate of growth of the tumour and of the development of metastases was very great. Nevertheless, at no stage when this case was under observation could any signs of hyperparathyroidism be demonstrated.

From the accepted accounts of the embryology of the parathyroid glands it is clear that a 'rest', or a complete arrested gland, might occur in an adult in the situation where this tumour arose.

It is well known that simple adenomata of the parathyroids are often accompanied by the clinical syndrome of hyperparathyroidism, the experimental study of which has been facilitated by the isolation of the active principle of the parathyroids by Collip.<sup>8</sup> Increased functional activity of the parathyroids, however produced, gives rise to depletion of bony calcium with consequent hypercalcaemia, excessive output of calcium in the urine, and gross changes in the bones.

In all the cases referred to in the literature the bony changes have been associated with simple adenomatous hyperplasia of the parathyroid glands. In no case of malignant disease of the parathyroids has the clinical syndrome associated with hyperparathyroidism been observed. The case under discussion has proved to be no exception to this rule, for an extensive X-ray examination of the skeleton which was carried out by Dr. F. Henderson revealed no obvious rarefaction of the bones. Moreover, repeated estimations of the blood calcium by Dr. A. S. McFarlane gave readings always below 10 per cent.

It is recognized that the histology of parathyroid tumours, and especially those having malignant characters, is very variable, resembling in some cases the hypernephromata, and in others carotid-body tumours. Dr. D. McLaren, in a private communication, pointed out the similarity of the cells of the tumour herein described to decidual cells, of which small groups are sometimes encountered as 'rests' in the adult. Histologically this tumour appears to us to resemble a malignant parathyroid rather than a carotid-body tumour or any other type mentioned. In this connection special attention is directed to the structure of the individual cells, to the presence of occasional eosinophilous cells, and, in one instance, to the finding of colloid-like material in the tumour.

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## THE OPERATIVE TREATMENT OF THE ELEPHANTOID SCROTUM.

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THERE are in the whole field of surgery few operations so successful, so simple, and so gratifying to both patient and surgeon as the radical cure of the elephantoid scrotum. It is in many ways a dramatic procedure. "In no other operation in surgery is so large a mass of solid tissue separated from the patient with complete success and little danger"<sup>1</sup>; and, apart from this consideration, the patient is rescued from a state of the profoundest shame and despair, due to his sexual impotence, the crippling influence of his tumour, and the frequent attacks of elephantoid fever which afflict him, and is converted into a normal human being capable of enjoying life and performing useful work in the world of men.

In view of these facts it is surprising to find that no adequate description of the operative treatment of scrotal elephantiasis exists in our leading surgical text-books, although these deal quite fully with what many consider to be the futile operations (of Kondoléon, Lanz, Rosenow, etc.) performed for elephantiasis of the limbs. It is no part of my present purpose to discuss the Kondoléon type of operation, but I feel compelled to state that, after quite a reasonable trial, I have abandoned it as useless and feel satisfied that any benefit which *seems* to follow it is really attributable to the rest in bed which it enforces. Vastly different are the results of the operation for scrotal elephantiasis.

There is a noticeable tendency amongst certain writers to magnify the difficulties and dangers of the operation. Manson-Bahr,<sup>2</sup> writing in an optimistic strain, hints at a mortality of 5 per cent. Chatterji<sup>3</sup> is particularly alarming, and his standard of treatment is one to which few isolated tropical surgeons will ever be likely to attain. He insists on the patient's being examined and sounded, physically, chemically, and bacteriologically, by almost every device of modern science, the central nervous system, the hepatic function, the sugar-tolerance, and the flora of the fæces and urine being all thoroughly investigated. He proscribes operation in the presence of oral sepsis; gonorrhœa or any of its complications; skin infections of any sort; and such constitutional diseases as diabetes, nephritis, chronic malaria, kala-azar, advanced cardiac or pulmonary diseases, arteriosclerosis, syphilis, and tuberculosis; although, surprisingly, he omits hookworm anæmia from his list of contra-indications. The patient, having been cured of these afflictions and thus rendered operable, has still a trying ordeal to face before coming under the knife. For at least a week he is kept supine, the scrotum being elevated and subjected to a daily course of washing and massage. He



is given frequent hot medicated hip-baths; he swallows calcium; and he suffers the following formidable series of injections—iodolysin, an autogenous vaccine prepared from the tissue fluids, pneumococcus vaccine to prevent post-operative pneumonia, morphine, atropine, and a solution of sodium bicarbonate and glucose in normal saline to prevent shock and acidosis. During the actual performance of the operation, the precautions taken against chill are so elaborate as to convince one that children operated on in Britain for intussusception or pyloric stenosis have hitherto been grossly neglected. A small army of three sisters and five other assistants (including one who is "tall, thin and muscular") aids the surgeon in his manipulations.

With all due respect for the opinion of a surgeon so accomplished and experienced as Chatterji, I feel satisfied that in this matter of the elephantoid scrotum he has made a mountain out of a mole-hill. I have personally found the operation to be simple and about as free from risk as the radical cure of an uncomplicated inguinal hernia.

**Criticism of Operative Methods.**—Two faults of technique are apparent in all the descriptions I have read of the operation for serotal elephantiasis: (1) The question of hæmostasis is either begged or else inadequately dealt with; and (2) The reflection of skin-flaps is postponed until *after* the amputation of the tumour—a procedure which means either leaving some of the elephantoid tissue unexcised, or else redividing, during a final dissection of the perineum, vessels which have been already cut.

One cannot too strongly condemn MacLeod's method<sup>4</sup> of attempting to control hæmorrhage by means of a rubber tourniquet wound round the 'neck' of the tumour and also round the pelvis. Such a tourniquet introduces an added risk of sepsis; divided tissues retract above it and then bleed vigorously; it so goffers the skin that one is unable either to mark out or to raise skin-flaps while it is in position; it cannot act as an efficient hæmostat without at the same time exerting injurious pressure on the cords, penis, and urethra (and perhaps on an unrecognized hernia); and when finally it slips or is removed a great deal of blood is lost before the ensuing hæmorrhage can be adequately controlled with pressure forceps. Theoretically, MacLeod's tourniquet is unsound, because it constricts the tissues below the level at which they should be divided. In order to lay bare the perineal muscles (in my opinion an essential part of the operation), the tissues must be cut well above the 'neck' of the tumour.

Most operators now work without a tourniquet, but they have provided no efficient substitute for it. With trifling differences in unimportant details, the operation as commonly performed proceeds as follows: First, the future skin-flaps are outlined *but not raised* (a process which in itself leads to a very persistent oozing from divided skin veins). Next, the penis and spermatic cords are dissected free and liberated, usually by means of three long vertical incisions, sometimes through a single median vertical incision,<sup>5</sup> and are then wrapped in gauze and placed on the lower abdomen. (The hæmorrhage from these vertical incisions is always severe and may be alarming, and owing to the yielding nature of the scrotal tissues and the fact that many of the large divided vessels are prevented by fibrous tissue from retracting, it cannot be adequately controlled by gauze packing.) The tumour mass is now amputated

at the level of the unreflected skin-flaps, and as each vessel has to be secured both proximally and distally, an enormous number of pressure forceps is required. The skin-flaps are now reflected right to the inner aspect of the thighs (with resultant further hæmorrhage), and as a rule the perineum is dissected free from the remnants of the elephantoid tissue (which means that there is still further bleeding and that the same vessels have been divided twice). The skin-flaps are now brought together over the cords and testicles, and the penis is dealt with by one or other of several available methods. Some operators clothe the penis with the preserved inner lining of the hypertrophied prepuce (a very good method when available); others utilize a suprapubic flap of skin (undesirable because of its hairiness); while yet others advocate Thiersch's method of skin-grafting, either immediately or at a later date (although confessing that the resultant covering is too thin and is insufficiently mobile).

In a modification of the foregoing method<sup>6</sup> the operation commences with the liberation of the penis, cords, and testes through a single median vertical incision which is then continued so as to split the entire scrotum into halves, each of which is then secured at its base by a large clamp. Everything distal to the two clamps is now severed. All vessels projecting beyond the clamps are ligated. The skin-flaps are then raised and the operation is concluded in the usual way. (By this method, unfortunately, one locks the stable-door after the horse has gone. The liberation of the penis, cords, and testes is effected before any means have been provided to render the dissection bloodless; and it is precisely the liberation of these structures which is responsible for nearly all the hæmorrhage encountered in the amputation of an elephantoid scrotum.)

### THE WRITER'S METHOD.

I have but little to say on the question of pre-operative treatment. The elaborate precautions advocated by Chatterji and others may perhaps be theoretically excellent, but in practice they can seldom be taken. The African native is inclined to be both timid and impatient, and, having screwed up his courage to the point of entrusting himself to a European surgeon, he wants to get his ordeal over and finished with as soon as possible. If ordered an elaborate course of pre-operative treatment, it is practically certain that he will never get as far as the anæsthetist, but will abscond and return to his village despondent and convinced that his infirmity is a matter of *shauri mungu* (God's will) and is incurable. Consequently, I have never yet refused operation, no matter how ill or senile the patient, except in cases of mortal illness or of advanced hookworm disease; and I have always endeavoured to operate within a few days of the patient's arrival in hospital. *Safura* (hookworm disease) is so well recognized and dreaded by the natives that they can usually be made to appreciate the necessity for having it treated before an operation is performed.

My pre-operative treatment consists merely in rest, with elevation of the scrotum, which is daily scrubbed with ether soap and water.

At the actual operation the patient is placed in the lithotomy position,

with the serotum hanging over the end of the table. After a final scrubbing with soap and water, the affected parts are etherized and iodined and towels are suitably arranged. A circular incision is then made right round the 'neck' of the tumour, dividing only skin plus a very small quantity of subcutaneous tissue. A large amputating knife is then thrust proximally under the skin till its point reaches the vicinity of one external abdominal ring. With a rapid sawing movement the knife is then carried across, subcutaneously, till its point reaches the opposite ring. An assistant meanwhile keeps packing

gauze beneath the skin in the wake of the advancing knife, and by exercising pressure over the flap reduces bleeding to a minimum. (*Fig. 501.*)

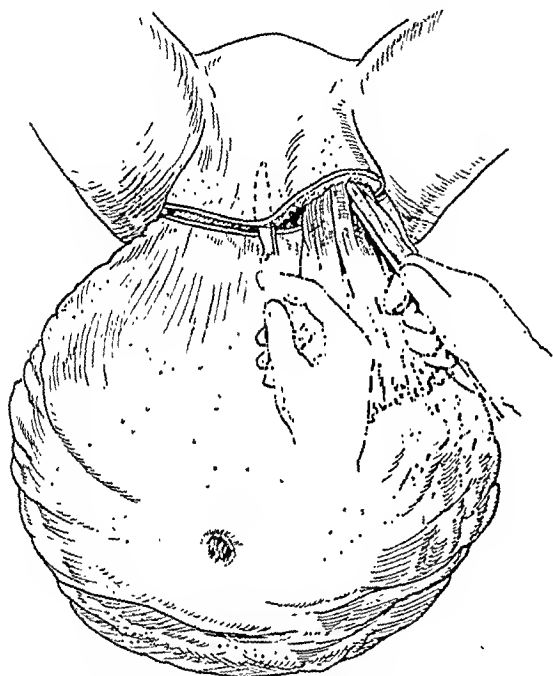


FIG. 501.—Undermining of the skin 'collar'.

In this way one half of a collar-like skin-flap is fashioned. With the help of a forehead lamp all bleeding-points beneath the flap are then secured in pressure forceps, the gauze packing being removed bit by bit while this is accomplished. The lower half of the 'collar' is now freed in a similar way, the point of the amputating knife being held close against the perineum and the adductor origins while the undercutting is performed. Hæmostasis is effected as before. Persistent bleeding points on the distal skin edge are secured by means of Ballance's scalp-clamps.

I would here emphasize that it is only during the preparation of my skin-flap or 'collar' that I encounter any hæmorrhage worth considering, the remainder of the operation being practically bloodless. The amount of blood lost is trifling and cannot be compared with that lost during the undermining of the skin at the commencement of a breast amputation.

I always prepare a 'collar' which will be large enough to cover completely and *without tension* both cords and testes. Consequently it often happens that the 'collar' is largely composed of more or less elephantoid skin. This is a matter of no moment, because the operation opens up several new channels for lymph drainage, as the result of which the skin eventually loses its elephantoid character.

The skin 'collar' now reflected, a one-inch incision is made over the upper part of each spermatic cord, near the external abdominal ring, and also over the root of the penis near the suspensory ligament. First a forefinger and then a gauze loop is passed round each of the three structures thus exposed and isolated at their base (*Fig. 502*).

The point of a large clamp is now pressed beneath the isolated segment of penis and is forced backwards through the entire thickness of the tumour, which is thus separated into halves. Each half is then grasped, about one inch distally to the pubes and perineum, by a powerful clamp, the cords and penis being carefully avoided. One clamp is closed. The scrotum is then elevated for a few minutes, to let some of the blood drain out of it. The second clamp is then closed and the scrotum allowed to resume its pendent position.

The penis and both cords and testes are now fully exposed through long vertical incisions (*Fig. 503*) and are carefully

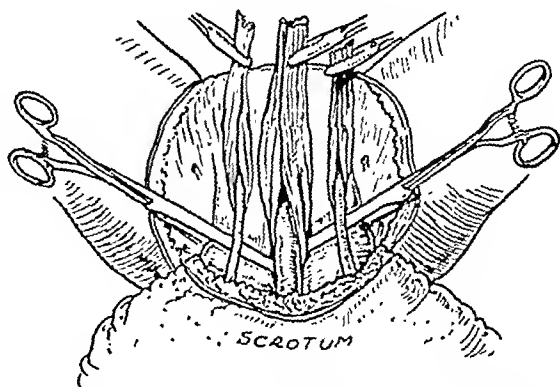


FIG. 502.—Isolation of the roots of the spermatic cords and penis, and clamping of the tumour 'neck'. Sometimes, instead of using clamps, the writer strangles the 'neck' with a couple of strong silver wires.

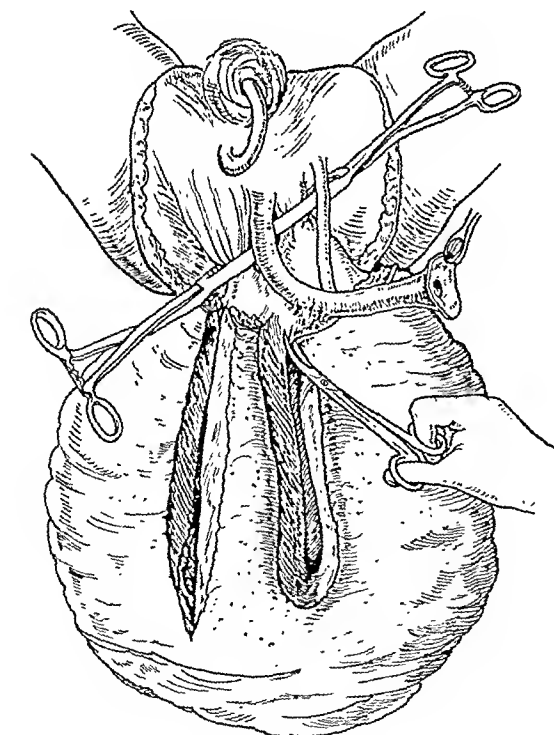


FIG. 503.—Freeing of the spermatic cords, testes, and penis through three large vertical incisions.

this procedure being absolutely bloodless owing to the hæmostatic action of the two large clamps. The greatly hypertrophied gubernacula are divided by scissor-snips. By means of a circular incision around the preputial opening, as much foreskin as possible is saved, so that it may subsequently be used as a covering for the penis.

What I consider to be a very important step in the operation is now performed—namely, the establishment of a direct lymph pathway from what will be the new scrotum to the lumbar glands. To effect this, the fibrous coverings of both spermatic cords are completely excised till the contained blood-vessels are clearly seen.

At this stage a hydrocele or hernia or both have frequently to be dealt with on one or both sides. The hydroceles are treated by the most rapid method avail-

able—usually by eversion, with or without partial excision of the sac; while atrophy of a testis may call for orchidectomy. Hernias are best

treated in a purely temporary manner by simple ligation of the sac as high as possible, without the external oblique being divided or the inguinal

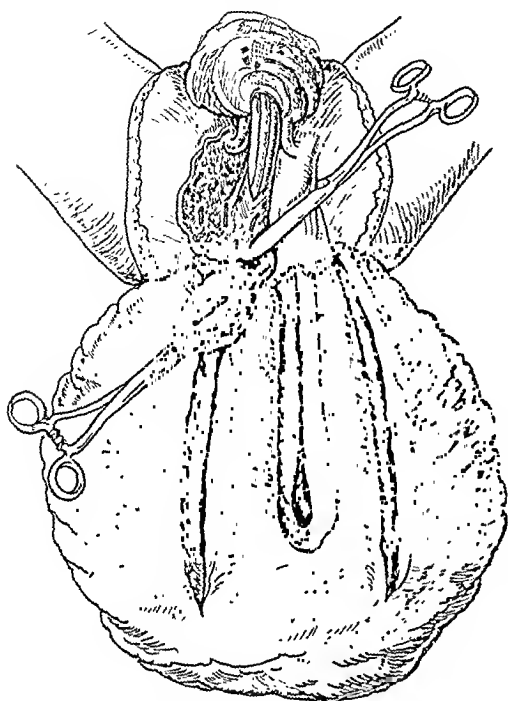


FIG. 504.—Amputation of the elephantoid mass.

canal subjected to any process of plastic repair. The reason for this is that should sepsis ensue (as is not unlikely), the safety of any plastic hernial operation would be gravely jeopardized. The hernia should be radically treated some weeks later.

With the hope of opening up a direct lymph pathway via the internal pudic and prostatic lymphatics to the internal iliac glands, great care is taken to lay bare the perineal muscles and divide the line of fusion between Colles's fascia and the two layers of the triangular ligament.

The penis, cords, and testes are now wrapped in sterile gauze and placed on the lower abdomen out of harm's way.

All vessels in the grasp of pressure forceps are now conscientiously ligatured, hæmatoma formation being one of the bugbears of the operation. The whole raw area is refreshed with application of hot saline, and the edges of the skin 'collar' are united along a median vertical line in such a way as to form a new scrotum into which the cords and testes are snugly tucked. A small rubber tube is inserted through the lowest end of the suture-line and will be retained for forty-eight hours. The penis is now covered by pulling back over it, like the finger of a glove, the preserved inner lining of the prepuce, the cut edges of which are fixed by

The amputation of the elephantoid scrotum is now completed by a series of short scissor-snips and knife-cuts *proximal* to the two large clamps (Fig. 504). Bleeding vessels are secured by forceps as they appear, and as we are here dealing with main trunks, the total number to be secured is not great. Moreover, as the *distal* ends of the vessels do not need attention, not more than a dozen Spencer-Wells forceps will be required.

FIG. 505.—Completion of the operation. The cords and testes have been covered by means of a vertical suture of the edges of the skin 'collar'. The penis has been covered by suture of its preserved and fully retracted prepuce to the edges of the upper part of the skin 'collar'. A small rubber drainage tube has been inserted through the lowest angle of the wound.

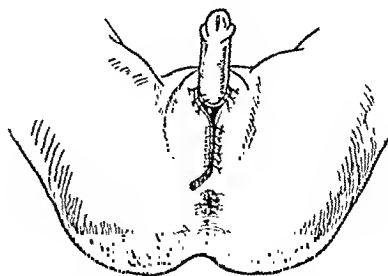


FIG. 505.—Completion of the operation. The cords and testes have been covered by means of a vertical suture of the edges of the skin 'collar'. The penis has been covered by suture of its preserved and fully retracted prepuce to the edges of the upper part of the skin 'collar'. A small rubber drainage tube has been inserted through the lowest angle of the wound.

a few points of suture to the edges of the uppermost portion of the skin 'collar'. (Fig. 505.)

Where, for one reason or another, sufficient healthy foreskin cannot be conserved to act as a penile covering, I have several times left the penis raw



FIG. 506.—The writer's 'tunnel' operation used in cases where the prepuce is too diseased to be available as a covering for the denuded penis. The photograph was taken ten days after operation.

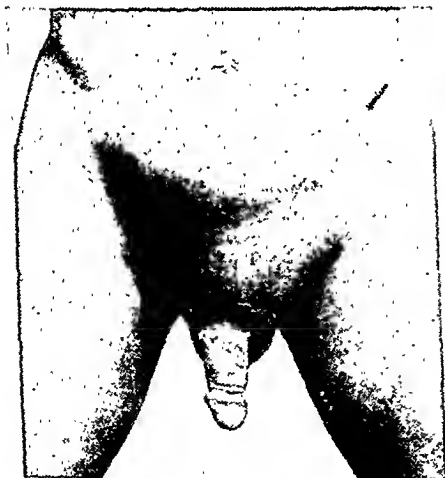


FIG. 508.—Case complicated by a large 'sliding' hernia on the right side. The hernia was treated first (in October, 1930), appendectomy being performed at the same time. The scrotum was amputated a fortnight later. The photograph was taken in June, 1931.



FIG. 507.—A very typical elephantoid scrotum. Weight after removal, 45 lb. No hernia present. Operation is much simplified when the tumour 'neck' is narrower than in this case.

and successfully clothed it at a later date by means of a granulating flap cut from the thigh and applied like a roller-bandage to the penis.<sup>7</sup> At other times I have threaded the raw penis temporarily through a subcutaneous tunnel (Fig. 506) on the inner aspect of the thigh and liberated it some ten days later by means of a simple flap incision.<sup>7</sup> Thiersch's method appears to be unsuitable, and I have never used it.

**After-treatment.**—Saline transfusion is rarely necessary and is never given as a routine. Morphia is administered. Catheterization is seldom

called for, probably because no bougie is used as a guide to the urethra in my operation. The wound is inspected daily because it has been my experience that a certain mild degree of sloughing is almost inevitable. When sloughing is at all marked, eusol dressings are employed.

**Mortality.**—In a series of 56 cases operated on between September, 1927, and July, 1931, there has been but one death. This was due to spreading cellulitis and septicæmia, and occurred in a very old and debilitated patient.

**Results.**—Nothing is more difficult than to trace the end-results of operations on African natives; but this much can be said: I have never yet seen a recurrence in any case of elephantoid scrotum operated on by me. On the other hand, of the not inconsiderable number of patients seen at intervals varying from a few months to three years after operation, *all* have been in an entirely satisfactory condition. The new scrotum has shown a marked tendency to become more and more supple with the lapse of time, while the physical and mental improvement in the patients has been striking. Two cases are illustrated in *Figs. 507, 508.*

### SUMMARY.

1. The operative treatment of scrotal elephantiasis is one of the most satisfactory procedures in surgery.

2. The view is advanced that the success of the operation depends on the establishment of new lymph pathways—namely, through the internal pudic and the prostatic lymphatics to the internal iliac glands and through the spermatic lymphatics to the lumbar glands. In order to open up these new pathways effectively, it is necessary to excise the fibrous coverings of the spermatic cords and to lay bare the perineal muscles.

3. The failure of operations for elephantiasis of the limbs is due to the impossibility of establishing a new lymph pathway in such cases.

4. The customary operations for elephantoid scrotum are criticized. It is thought that they do not provide sufficiently for hæmostasis or for the establishment of new lymph pathways; and it is considered that the practice of raising the skin-flaps *after* the amputation of the scrotum is unsound.

5. The writer's method of operating is described.

6. The danger of the operation is extremely slight.

I have to thank Dr. A. H. Owen, Acting Director of Medical and Sanitary Services, Tanganyika Territory, for permission to submit this article for publication.

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## *SHORT NOTES OF RARE OR OBSCURE CASES*

### NOTE ON A CASE OF PITUITARY TUMOUR TREATED BY RADON SEEDS.

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IN a previous number of the *BRITISH JOURNAL OF SURGERY*,<sup>1</sup> I contributed towards the surgery of the pituitary region by reporting on four cases where treatment had been carried out by the introduction of radon seeds into the tumour mass, the seeds varying in number from 14 to 8. In the summary it was stated: (1) That radon seeds could be introduced into, and spaced throughout, a tumour of the pituitary region, without actually exposing the



FIG. 509.—Antero-posterior view, showing the correct spacing of the six seeds.



FIG. 510.—Lateral view, showing the six seeds in the lower and posterior part of the tumour.

tumour to view; (2) That neighbouring structures could be avoided; and (3) That the results obtained were not discouraging. A fifth case, operated on in July, 1931, enables me to confirm the first of these three statements, leaving the second and third requiring some amendment.

The operation was conducted in the same manner as before—right frontal bone-flap, dura and brain elevated with my special illuminating retractor, as far back as the posterior border of the anterior fossa, and a small incision made through the dura mater, close up against the anterior clinoid process, through which the introducer was inserted. No attempt was made to expose the tumour itself; I relied on my experience of the direction required. Six



seeds were introduced, with spacing, and I estimated that they were placed in the posterior half of the tumour. Desiring to place more seeds in the anterior part of the tumour, the introducer was re-inserted. This was at once followed by the escape of blood along the cannula, which, with the sudden swelling up and discoloration of the dura, showed that I had injured some vessel. The patient was sent back to bed, and died two days later with symptoms of compression.

The post-mortem showed: (1) A tumour (adenoma), pear-shaped and plum-coloured, seated in the pituitary fossa, wholly occupying that region; (2) Some blood between the dura and the brain, but less than was expected, and no evidence of injury to any special blood-vessel; (3) No sign on the surface of the tumour to show where the introducer had entered.

The whole region was cut away, with the underlying portion of the base of the skull, hardened, and X-rayed (*Figs. 509, 510*). The photograph shows that all six seeds are well placed and spaced in the posterior part of the tumour. With a little more luck, the second series of six seeds would have been inserted into the anterior half.

It is possible that the vascular injury might have been avoided if the tumour had been exposed, but this would have been very difficult, and certainly very dangerous.

As against this unfortunate result, I have to report that the very successful case recorded in my series remains perfectly well, with full working capacity.

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#### REFERENCE.

<sup>1</sup>*Brit. Jour. Surg.*, 1931, xix, 68.

## REVIEWS AND NOTICES OF BOOKS.

*Diseases of the Thyroid Gland: with Special Reference to Thyrotoxicosis.* By CECIL A. JOLL, M.S., B.Sc. (Lond.), F.R.C.S., Senior Surgeon to the Royal Free Hospital and the Miller General Hospital, etc. Crown 4to. Pp. 682 + xviii, with 283 illustrations and 24 plates. 1932. London: William Heinemann (Medical Books) Ltd. 63s. net.

THE author states in the preface that it is thirty years since a book on the diseases of the thyroid gland and their treatment has been published in Great Britain. During that period the advance in knowledge of this subject has been very great. Workers in many countries have contributed to that advancement, and the contribution from the British Empire having been considerable, it was high time that an adequate presentation of the subject should be made in Great Britain. It may be said at once that the book has been worth waiting for, Mr. Joll having envisaged a work to include every aspect—"a general survey"—of the subject. The task for one individual was big, and for a surgeon occupied with the duties of a teaching hospital and private practice it was Herculean. Mr. Joll has inherited much from a hospital where Sir James Berry had already achieved a world-wide reputation in this subject; how widely he has read is seen from the ably selected references, and his own work over many years has given him first-hand knowledge of pathology and the surgical problems. The judgements are balanced, and mature experience is apparent throughout. This does not necessarily mean that every conclusion will be accepted. No book can pronounce the final word, for in many of the problems there is not yet unanimity, and in a subject where so many workers are giving their lives to one or other of the aspects of the subject, new material becomes available between the time a book of this magnitude goes to press and that of its publication.

It is fitting that the frontispiece should be an engraving of Caleb Hillier Parry. Too often in foreign literature the honour of having first described accurately the symptoms and signs of toxic goitre is wrongly ascribed elsewhere, but it must be remembered that although his case histories and conclusions were written out with great care, Parry did not publish them.

The chapters on development, structure, and physiology of the thyroid gland are very complete, and contain adequate references to the work of Gale Wilson, Rienhoff, Kendall, and Harington. The chapter on the parathyroid glandules, while complete in every other respect, does not contain reference to the work done abroad and in Great Britain on parathyroid tumours associated with generalized fibrocystic disease of the bones.

It is a great satisfaction to practitioner and student to see the classification of diseases of the thyroid gland becoming more simple. As is well known, this author separates Riedel's disease from lymphadenoid goitre, thus differing from Ewing and some others. The illustrations and descriptions of these conditions are excellent. The term 'parenchymatous' is given its proper significance. It has too long been employed in England to denote diffuse colloid goitre. Primary malignant epithelial tumours are described under three well recognized types, and one wonders if it is necessary or wise to give a separate heading in the classification to 'mixed-celled' types—carcinoma-sarcomatodes and sarcoma-carcinomatodes. The author points out that Kettle and Ewing have described the metaplasia occurring in these tumours, which are undoubtedly epithelial in origin.

The chapters dealing with the etiology, distribution, and prevalence of simple goitre are exhaustive, and the immense amount of work done on these subjects in recent years has been critically reviewed. Possibly the most contentious chapter

will be that in which the evolution and histopathology of diffusely nodular goitre is discussed. Where so many Continental and American pathologists who have been investigating this for decades—and are still doing so—fail to reach a conclusion, Mr. Joll's marshalling of evidence, masterly as it is, may well leave the reader in doubt. He believes that there is no essential difference between the colloid and solid forms of nodule, and he retains the term 'adenomatosis' to describe the common form of goitre associated with multiple colloid nodules in this country. Reinlioff and others have regarded these as purely involution nodules. Marine, in the article from which Mr. Joll quotes, admits that knowledge of these nodules is at present very inadequate, so that this chapter may be taken as a statement of the available knowledge at the moment.

Nearly half the book is devoted to thyrotoxicosis. Pathogenesis, signs, symptoms, metabolism, and treatment are dealt with in great detail. The increase in the death-rate from the disease—taken from official figures—during the last five years is striking. The author ascribes this to the almost universal use of iodine in prophylaxis and treatment. One wonders if it may not in part be due to the greatly increased number of operations performed throughout the land. The margin of safety in this operation is still a narrow one, requiring careful selection of cases and of the time of operation, long preparatory treatment, and considerable skill on the part of the surgeon. Mr. Joll takes the increase in the death-rate to indicate that there is a still greater proportional increase in the prevalence of the disease.

In accord with the growing custom in this country Mr. Joll divides cases into primary and secondary thyrotoxicosis rather than into exophthalmic goitre and toxic adenoma. The distinction is that in secondary toxic goitre the toxic condition is "engrafted on a pre-existing goitre". He states that he has had patients in whom the toxic symptoms supervened on goitres primarily of the diffuse symmetrical colloid type. Surely the case illustrated in *Figs. 201, 202, and Plate XXXIII*, belongs to this class. The very big goitre with large colloid vesicles implies that a colloid goitre preceded the toxic condition.

The special clinical groups—those occurring in childhood or old age, in association with pregnancy, with delirium, or other mental disorders, with cardiac complications, with diabetes, or with abdominal crises—are discussed.

X-ray and radium treatment is reviewed. The author does not favour the use of iodine in the medical treatment of the disease, but would reserve it for the period immediately preceding operation. Physicians will scarcely agree with this view, but there is no doubt that its use is often not sufficiently controlled. A full and most helpful chapter is devoted to anaesthesia in all its modern aspects. Pre-operative and post-operative treatment is adequately dealt with. It is pointed out that operation is unjustifiable in hyperacute cases, or during the exacerbations of the disease.

Possibly a little more space might have been given to the medical preparation and after-treatment of patients with auricular fibrillation and heart failure. These patients are often oedematous to the waist when first seen, long and skilled treatment being required to render them fit for operation. In the three cases illustrating operation in this condition normal rhythm returned spontaneously. This occurs in nearly half the cases operated upon, but readers would have welcomed more detailed help in the subsequent treatment of the others. The author gives the credit for popularizing the extensive bilateral operation to British surgery—a fact, as he states, not always recognized in America. Regarding the operation of thyroidectomy, no detail is left to the imagination, each step being minutely described and illustrated. Following this, the dangers and difficulties which may be met with are discussed, and treatment is indicated for the post-operative complications. Each surgeon will develop his own technique, and will differ in some respects from others. Mr. Joll has described a good operation, though he has not made it appear simple. This is wise, for even in a moderately severe case of this disease the operation is not to be undertaken without due consideration and careful preparation.

Mr. Joll has presented his fellow-practitioners and students with a monumental volume. They need not trouble to search the literature published up to the time

this book went to press, for they will find everything relevant within its covers. They will get plenty of practice in sifting evidence—they may even wish that some of the more obsolete references had been omitted—but when they have read it they will know all that was known on the subject of goitre in 1931.

Mr. Joll is to be complimented on the illustrations and diagrams, and the publishers on producing a book that will take a very high place in British medical literature. (The word *resected*, page 623, four lines from the bottom, is probably a misprint, and, if so, is the only one discovered in the book.)

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**Thomson and Miles' Manual of Surgery.** By ALEXANDER MILES, M.D., LL.D., F.R.C.S.E., Consulting Surgeon, Royal Infirmary, Edinburgh; and D. P. D. WILKIE, M.D., F.R.C.S.E., Professor of Surgery, University of Edinburgh. Eighth edition. Crown 8vo. Vol. II. Extremities, Head and Neck. Pp. 685 + viii, with 303 illustrations. Vol. III. Thorax and Abdomen. Pp. 578 + xii, with 177 illustrations. 1931. London: Humphrey Milford. 12s. 6d. net each.

THROUGHOUT these volumes there are many evidences of careful revision in order to make the work a comprehensive and up-to-date record of surgical teaching in Edinburgh. This applies especially to the chapters on the thyroid gland, the chest, the gall-bladder, and the breast; and to the description of the investigation of a urinary case, all of which have been largely rewritten. Accounts of the modern conception of the gliomas, and of diverticulitis of the colon, and fuller references to radium treatment are valuable additions.

In previous editions there had been a tendency to illustrate the late stages of disease and pathological curiosities. It is therefore a pleasure to note that the pictures also have been considerably revised, those of advanced disease being replaced by skiagrams of barium examinations, pyelograms, and other illustrations of diagnostic methods. That the old passion for freaks is not yet conquered is shown by the inclusion of the useless though amusing skiagram of a hair-ball in the stomach, and the photograph of an 'enterolith' of heather-roots and twigs!

It should be our concern to assess the value of the work as a whole rather than to criticize matters of detail, yet we cannot but note in a book whose standard is generally so high the occurrence of certain features which seem to have escaped attention. Misleading or even inaccurate statements occur in connection with intracranial hæmorrhage, particularly regarding traumatic apoplexy and subdural hæmorrhage. It seems a mistake to adduce the absence of glandular involvement as evidence against malignancy in the case of a doubtful lesion; and the view that the mixed salivary-gland tumours develop from rests derived from the first branchial arch is surely obsolete. Finally, it is a surprise to find, beside an illustration of a tuberculous nodule in the globus minor of the epididymis, the statement that it is more than doubtful if the disease is ever due to a downward spread of infection from the prostate.

This manual gives on the whole an excellent description of the clinical and pathological aspects of surgical diseases, but little advice is offered regarding their treatment. It has previously been criticized on this account, and the authors probably have good reasons for not attempting to remedy the defect. It does seem a pity, however, that a manual running into three volumes should not be an adequate guide to treatment.

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**Pye's Surgical Handicraft.** Edited by H. W. CARSON, F.R.C.S., Late Senior Surgeon, Prince of Wales's General Hospital, Tottenham. Tenth edition, fully revised. Demy 8vo. Pp. 641 + xviii, with 343 illustrations and 22 plates. 1931. Bristol: John Wright & Sons Ltd. 21s. net.

THIS book is too well known to require much added praise. This, the tenth edition, has been brought up to date by the late Mr. Herbert Carson, who died when only two sheets of the edition remained to be seen through the press. The book has been increased in size by only a few pages, but many sections have been revised and some rewritten, and fresh sections have been added on a few subjects, such as the

tannic acid treatment of burns, the injection treatment of varicose veins, etc. We would like to see more revision in the chapter on fractures, bringing the treatment more into line with modern methods. In describing the treatment of intracapsular fracture of the femoral neck it is advised that "the patient should be propped up in bed with the limb placed between sandbags". After about a month the patient is advised to use the limb as much as possible "since union is not aimed at". This surely is not the best advice for house surgeons and dressers.

Apart from a small number of facts which are still somewhat behind the times, this book stands out as probably the best of those on minor surgery and its craft, as indeed it has done for the last forty-seven years; and it can confidently be recommended to house surgeons and dressers as well as to practitioners as giving a good account of the many small points in all forms of minor surgery which are so difficult to carry in one's head.

*La Pratique chirurgicale illustrée.* By VICTOR PAUCHET. Fasc. XVII. Super royal 8vo. Pp. 268 + vi, with 186 illustrations by S. Depret. 1932. Paris: Doin et Cie. Fr. 65.

IN this fasciculus Victor Pauchet, while contributing most of the articles himself, has included articles by five other surgeons, and the volume opens with a description by de Martel of a new method of craniectomy for exposing the cerebellum—a method which gives a beautiful exposure for dealing with acoustic tumours, meningiomas in the region of the lateral sinus, or tumours of the cerebellum and fourth ventricle. Dr. Bonnet Roy writes on the radical cure of dental cysts, and Dr. A. Jung on Matas' operation for obliteration of popliteal aneurysm. Two articles on plastic surgery are included, one for depressed nose by Dr. J. Bourguet, and the other for hypospadias by Dr. le Gac. In the former we seem to recognize the work of Sir Harold Gillies, and the latter illustrates the method of Ombredanne—a most ingenious operation very well illustrated. We have commented before on the excellent value of M. Depret's illustrations, which often render the text unnecessary. It would be extremely difficult to give a lucid description of this operation for hypospadias; the illustrations supplied require no verbal explanation.

Pauchet writes on a variety of subjects, which include operations for cancer of the breast, hernia of the lung, biliary fistula, anastomosis of ureter and colon, and gastric ulcer penetrating the pancreas. In these he maintains his usual high standard. With regard to the article on diverticulitis, while agreeing that the majority of cases should be treated on medical lines, our operative experience makes us more than sceptical of the practicability of multiple diverticulectomy, i.e., resection of individual diverticula (except in isolated instances), and we regard the operation described as unpractical if not valueless.

It is to be hoped that some day Pauchet will collect the articles which deal with the different branches of surgery and publish them in separate volumes. In these days of specialism a mixed grill does not attract the average reader as perhaps it should.

*Emergency Surgery.* By HAMILTON BAILEY, F.R.C.S., Surgeon, Royal Northern Hospital, London. Vol. II. Thorax, Spine, Head, Neck, Extremities, etc. Large 8vo. Pp. 415 + xviii, with 430 illustrations, some in colour. 1931. Bristol: John Wright & Sons Ltd. 25s. net.

THE first volume of this work on *Emergency Surgery*, that dealing with the abdomen and pelvis only, was reviewed in the April, 1931, number of this JOURNAL: the present volume covers the remaining field. The high quality of its predecessor has been fully maintained in the present volume, and the two together form a most valuable and reliable guide to the occasional surgeon and to the "comparatively isolated surgeon" when faced with a surgical emergency. Opinions must inevitably differ as to what exactly should be included in a work of this scope; but, on the whole, we consider that a very judicious selection has been made, and the writer, moreover, has not confused the issue by the too frequent insertion of alternative

methods. The text is certainly up to date, as evidenced by the full account given of Professor Meyer's technique for performing Trendelenburg's operation for the extraction of emboli from the pulmonary artery, the Winnett-Orr treatment of compound fractures and acute osteomyelitis, and the tannic acid treatment for burns. Blood transfusion is very fully described, and for emergency tracheotomy K. H. Digby's method of performing the operation is rightly recommended. In the special branches the writer has sought the assistance of experts, notably that of Mr. Eric Watson-Williams, who is responsible for the sections dealing with the nose, ear, and larynx, and with intracranial suppuration.

The illustrations are plentiful and excellent, and special mention must be made of the coloured one upon page 19, depicting traumatic asphyxia.

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**A Textbook on Surgical Pathology.** By CHARLES F. W. ILLINGWORTH, M.D., F.R.C.S.E., formerly Tutor in Clinical Surgery, Royal Infirmary, Edinburgh; and BRUCE M. DICK, M.B., F.R.C.S.E., formerly Tutor in Clinical Surgery, Royal Infirmary, Edinburgh. Royal 8vo. Pp. 677 + viii, with 290 illustrations. 1932. London: J. & A. Churchill. 36s. net.

THE criticism of a work on surgical pathology must be influenced by one's opinion upon the part such books of applied science have to play in education, and by what the author intends the book to accomplish. A pathologist, if he expects an exhaustive dissertation upon the purely pathological features of the diseases deemed to be surgical, is bound to find that some of the subjects in which he is interested are treated briefly, superficially, and with what he may consider scant respect. The clinician, if he looks for a decisive judgement upon the many unsolved problems of pathology, will also be disappointed. Criticism of this book from such extreme points of view would be unjust, for it has not been the intention of the authors to add anything to the science, but rather to stress features in the study of pathology which are of greatest value to the surgeon, and in this they may justly claim that they have been successful.

The size of the book has been wisely limited by the exclusion of detailed accounts of inflammation and repair, since it has been written for Final M.B. and Fellowship Students, and a knowledge of the fundamental processes is assumed. The early chapters deal with some of the problems of general pathology—shock, the general infections, and tumours—and the remainder with tissue and regional pathology, including the female generative organs. In all sections the morbid anatomy is adequately described, but the most valuable feature is the consideration of the nature of the conditions described, and the account, supplemented by ample references to recent literature, of experimental and clinical research upon which modern conceptions of disease are based. The illustrations are good, the microphotographs being particularly praiseworthy.

It is well-nigh impossible for an individual to keep abreast of the recent work in anatomy, physiology, and pathology which has a bearing on surgery; and the authors have the advantage of having been brought up in surroundings in which every encouragement is given not only to trial by experiment and to wide reading, but also to free and frequent interchange of ideas and knowledge among the group of men working in the Department of Surgery. This has enabled them to cover a wide field, but it is only to be expected that some chapters will carry more conviction than others, and it is not difficult to distinguish the subjects which have been investigated by the authors and their colleagues from those about which they have had to obtain the information by referring to the work of others.

The conscientious author may rightly hesitate to draw conclusions from observations not made by himself; but his writings will be of much greater value to the student, who is even less capable of discriminating between the various theories offered in explanation of a given phenomenon, if in addition to stating the theories, he indicates clearly which view has most to support it, and is of the greatest importance as a guide in the practice of surgery. If this more dogmatic attitude were adopted in parts of this book it would be of greater value in

teaching, even though some of the opinions expressed might require modification in future editions.

Most of the book is so good that it is disappointing to find a poor description of the pyogenic infections of bone, an inadequate account of injuries to the brain, and some rather confusing statements about hydronephrosis and renal infection. Its faults, however, are neither numerous nor serious enough to detract appreciably from the value of a book which is likely to prove a popular text-book for students and to provide for teachers a useful guide to the recent literature of surgical pathology.

**Surgical Pathology of the Genito-urinary Organs.** By ARTHUR E. HERTZLER, M.D., Professor of Surgery, University of Kansas. Hertzler's Monographs on Surgical Pathology. Royal 8vo. Pp. 286 + xviii, with 222 illustrations. 1931. London: J. B. Lippincott Co. 21s. net.

This is one of a series of monographs on surgical pathology by Hertzler: it is a well printed book of some 285 pages, and the illustrations are uniformly good and are of help in elucidating the text.

The author's views on this section of surgical pathology are always interesting, sometimes original, and occasionally inaccurate. His style is almost startlingly different from that of the usual text-book of English medicine; he manages to extract a good deal of humour from the dry bones of pathology: the one adjective appropriate to the tone of this volume is 'snappy', and the effect on the whole, though occasionally irritating to the English reader, is to make the work very readable.

In writing of the pathology of the tuberculous kidney, he exclaims: "The owl to the moon doth complain, but to present a working outline for the clinic is no easy task"; one can fancy Sir James Paget lifting his eyebrows if he had lived to read such a sentence in a book on his favourite subject.

We consider the bibliography poor; the author seems to have very little knowledge of the best of the English, German, and French literature on this section of surgical pathology.

On the whole we can recommend this book to our readers as an attractively written but not very profound monograph.

**Clinical Examination and Surgical Diagnosis.** By FÉLIX LEJARS (Paris). Translated by HELEN C. SCOTT, M.R.C.S., L.R.C.P. Large 8vo. Pp. 872 + xi, with 1094 illustrations. 1931. London: Jonathan Cape Ltd. 50s. net.

THIS work on 'surgical craft', as the author expresses it, is in no way a book of reference, but is designed as a guide to diagnosis by a consideration of what conditions should be conjectured in a particular case and of how the patient should be examined.

The justification for this further work on a well-worn subject derives from: (1) The attention paid to local physical examination, and the detailed directions for its systematic performance: ancillary methods are deliberately given scant attention. (2) In striking contrast with many works on surgical diagnosis, this one is most readable, for Lejars has a rare knack for vivid clinical pictures. In this respect he has been fortunate in his translator, Miss Helen Scott. (3) The book is the product of French thought—the references are almost exclusively to Frenchmen—and will appeal to those who would like to know something of French surgical outlook.

The majority of the illustrations are from photographs of patients to show either pathological conditions or methods of physical examination. They display clearly what they are meant to show, but there is an inclination, which is perhaps inevitable, to illustrate advanced rather than early conditions.

# REVIEWS AND NOTICES OF BOOKS

667

**Fractures of the Jaws.** By ROBERT H. IVY, M.D., D.D.S., F.A.C.S., University of Pennsylvania; and LAWRENCE CURTIS, A.B., M.D., D.D.S., University of Pennsylvania. Medium 8vo. Pp. 180 + viii, with 177 illustrations. 1932. London: Henry Kimpton. 21s. net.

THE authors have produced a most creditable volume which fills a much-needed want in this country, where specialization is so limited. America has its oral surgeons in almost every city, but here fractured jaws form a 'no-man's land' between the general surgeon and the dental surgeon, with the result that there is often lack of co-operation in the treatment of these cases.

The Preface ably summarizes the position with regard to the general surgeon in stating that "as long as cases are admitted to his service in the hospital it is his responsibility to see that they receive proper treatment, either by himself or by someone delegated by him who understands the problem. The haphazard way in which these cases are frequently handled at present is often a cause of permanent crippling of the functions of the jaw, unless correction is undertaken by measures requiring many additional months of treatment."

The authors have gone out of their way to produce a book giving a technique which may be carried out by the general surgeon having little or no special dental experience. They have avoided all complicated dental splints requiring special laboratory experience and advocate the eyelet-wiring technique, which is certainly very simple. In dealing with fixation of the fragments emphasis is rightly laid upon the fallacy of any attempt being made to wire or plate the fragments, as nearly all fractures are compound in the mouth. In the same chapter reference is made to the necessity of removing teeth in the line of fracture, which is the accepted teaching in this country.

The chapter on non-union gives a very clear description of the present-day methods of bone-grafting, the authors favouring the iliac graft; but it is noted that they use the method of wiring the graft in position rather than dovetailing. The chapter on dietary management, by Clyde W. Scogin, D.D.S., gives very valuable information on this important phase of the treatment of these cases, and completes an extremely practical book, which is easy to read and well illustrated. The volume will add materially to our literature on the subject, and should certainly be consulted by all surgeons undertaking the treatment of these cases.

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**The Intervertebral Discs: Observations on their Normal and Morbid Anatomy in Relation to Certain Spinal Deformities.** By ORMOND A. BEADLE. Issued by the Medical Research Council. Royal 8vo. Pp. 79, with 47 illustrations. 1931. London: His Majesty's Stationery Office. 2s. net.

THIS is one of the first and most complete accounts in the English language of the anatomy and pathology of the intervertebral disc. The writer has worked in Professor Schmorl's Clinic at Dresden over a long period, and has studied carefully his unique collection of spinal columns, some of the lesions of which are well illustrated in this paper.

The work is an excellent epitome of Schmorl's teaching, with observations and comments by the author. It is of especial interest to anatomists and orthopaedic surgeons; the anatomy, histology, and mechanism of the intervertebral disc are clearly described; a rational explanation of 'vertebral epiphysitis' is offered, and degenerative changes in the disc as the result of increasing age and wear and tear of everyday life are suggested as the etiological factor in the group of conditions generically termed 'spondylitis'. French and American observers, especially the former, have not been slow in following the work of Schmorl; the valuable writings of Calvé and Gailand have apparently been overlooked in the bibliography of the present monograph.

The author is to be congratulated on his choice of subject and place for this study, and for its comprehensive presentation to British surgeons.

VOL. XIX.—NO. 76.



*Die Elektrochirurgie.* By Prof. Dr. FRANZ KEYSER (Berlin). Royal 4to. Pp. 238 + x, with 232 illustrations. 1931. Leipzig: Fischers Medizinische Buchhandlung. Paper covers, M. 52; bound, M. 56.

DIATHERMY plays an increasingly useful part in surgical technique, and it would not be unfair to say perhaps that its true value is only just beginning to dawn on surgeons. Immense improvement in technical appliances is going hand in hand with education in this matter, so that ultimately it must take a recognized position as a surgical instrument of precision superior to the knife in certain restricted fields.

Keyser's book goes very thoroughly into the electro-technical details of the diathermy machine and the factors which influence its effects on the tissues dealt with. The greater part of the book, however, is devoted to clinical details of cases illustrating the practical application of the various types of diathermy in general surgery. The clinical chapters are exceptionally well illustrated and give admirable records of the usefulness of diathermy in the treatment of advanced malignant disease.

To those who are seeking to keep abreast of this very definite advance in surgical technique we can recommend this volume.

*Der künstliche Pneumothorax.* By HANS ALEXANDER. Royal 8vo. Pp. 42, with 54 illustrations. 1931. Berlin: Julius Springer. RM. 7.50.

THIS small volume gives an excellent account of the treatment of pulmonary tuberculosis by artificial pneumothorax. The most common complication is the formation of exudates into the pleura. These occur in 25 per cent and are generally clear effusions which disappear without treatment, but a small proportion develop into tuberculous purulent effusions. Intestinal tuberculosis, spondylitis, bilateral renal tuberculosis, emphysema and asthma, severe nephritis and diabetes, and uncompensated heart failure are absolute contra-indications. Some consideration is given to the combination of pneumothorax and phrenicectomy, and intrapleural pneumolysis is very shortly discussed. The skiagrams of cases after thoracoplasty show the posterior ends of the ribs to have been divided too far forward, thereby diminishing the collapse obtainable by the operation.

*Indikation und Technik des künstlichen Pneumothorax.* By Dr. HEINRICH MAYRHOFFER (Vienna). Medium 8vo. Pp. 35 + vi, with 33 illustrations. 1931. Vienna: Julius Springer.

THIS is a small, relatively elementary, volume on pneumothorax treatment, illustrated by small line drawings. Indications include tuberculosis, pulmonary abscess, bronchiectasis, and for diagnostic purposes. Only tuberculosis is dealt with at any length, and under the headings of general indications, contra-indications, and hæmoptysis. It is stated that the treatment should be continued until the sputum has been free from bacilli for at least a year. Cauterization and phrenicectomy are discussed, especially in their bearing on pneumothorax.

The whole presentation is that of the usually accepted treatment of tuberculosis by pneumothorax, which is the object of the publication.

# BOOK NOTICES.

*[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.]*

**A Descriptive Atlas of Radiographs: An Aid to Modern Clinical Methods.** By A. P. BERTWISTLE, M.B., Ch.B., F.R.C.S.E. Second edition, revised and enlarged. Crown 4to. Pp. 552 + xxviii, with 767 illustrations. 1932. London: Henry Kimpton. 42s. net.

**Intracranial Pyogenic Diseases. A Pathological and Clinical Study of the Pathways of Infection from the Face, the Nasal and Paranasal Air-cavities.** By A. LOGAN TURNER, M.D., LL.D. Edin., Hon. F.R.C.P.E., F.R.S.E., F.R.C.S.E., Consulting Surgeon, Ear and Throat Department, Royal Infirmary of Edinburgh; and F. ESMOND REYNOLDS, M.D. Edin., D.T.M.&H. Camb., M.R.C.P.E., Lecturer on Neuropathology, University of Edinburgh. Royal 8vo. Pp. 271 + xx, with 82 illustrations. 1931. Edinburgh: Oliver & Boyd. 12s. 6d. net.

**Guy's Hospital Reports.** Edited by ARTHUR F. HURST, M.D. Vol. LXXXI (Vol. XI, Fourth Series), No. 4. October, 1931. Royal 8vo. Pp. 379-498. Illustrated. 1931. London: The Lancet Ltd. Annual subscription £2 2s. net; 12s. 6d. per issue.

**Diagnosis in Joint Disease: A Clinical and Pathological Study of Arthritis.** By NATHANIEL ALLISON, M.D., F.A.C.S., Professor of Chicago, etc.; and RALPH K. GHORMLEY, M.D., Associate in Medicine, Mayo Clinic, etc. Large 4to. Pp. 196 + xii. Illustrated in colour and in black-and-white. 1931. London: Humphrey Milford. 52s. 6d. net.

**St. Bartholomew's Hospital Reports.** Edited by Sir THOMAS HORDER, Bart., K.C.V.O., RONALD G. CANTI, WILFRED SHAW, CHARLES F. HARRIS, J. PATERSON ROSS, R. C. ELSMLIE, W. GIRLING BALL, and GEOFFREY EVANS. Vol. LXIV. Demy 8vo. Pp. 231 + xxv. Illustrated. 1931. London: John Murray. 21s. net.

**Radium and Cancer.** By H. S. SOUTTAR, C.B.E., M.D., M.Ch. (Oxon.), F.R.C.S., Surgeon to the London Hospital, etc. Pocket Monographs on Practical Medicine. Fcap. 8vo. Pp. 64 + viii, with diagrams. 1932. London: John Bale, Sons & Danielsson, Ltd. 2s. 6d. net.

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# INDEX TO VOLUME XIX

	PAGE		PAGE
<b>ABDOMEN</b> , peritonitis, direct ob-		Ancurysm, subclavian, associated with	
servations of	635	cervical rib	334
Aberrant thyroids, lateral	92	Angioma, cavernous, of arm	360
Abrodil in intravenous pyelography	624	Ankle, fractures at	78
Abscess formation in hernial sac	182, 185	Anna Kliniek voor Orthopædie, Leiden	324
— intraperitoneal	638	Anti-gas-gangrene serum in peritonitis	638
Acetabulum, fracture of, with traumatic		Appendicitis, acute, analysis of	2126
dislocation of hip	601, 603, 605	— cases	
Achard-Thiers symptom-complex	559, 563	— in old age	410
Adenocarcinoma of middle ear	60	Appendix cecoploica of sigmoid colon,	63
Adhesions, perigastric, complicating		torsion of	508
chronic follicular gastritis	43	ARMITAGE, GEORGE: The successful	
Adrenal precocity	558	removal of an osteoma of the	
— pseudo-hermaphroditism	557, 561	frontal sinus	158
— virilism and hirsutism	558, 562	Arris and Gale Lecture, Jan. 30, 1931,	
Adrenalectomy, unilateral, in cortical		by Donald Hunter	203
hyperplasia	559	Arthritis, tuberculous, of knee (see Knee)	83
Adreno-genital syndrome	557	Arthroplasty in tuberculosis of knee	505
— case reports	561	Arthrotomy, diagnostic, in tuberculosis	495
— classification	557	of knee	
— conclusions	557	Atelectasis, post-operative (see Lung,	
— histopathology	569	Post-operative Massive Collapse of)	
— operative interference in	558, 568	Atropine in general peritonitis	639 643
— results of operation	569	— to prevent post-operative atelectasis	406
— summary of case reports	560	Axillary glands, radium treatment of, in	
— unilateral adrenalectomy in	559	breast cancer	420, 437, 452
Age distribution of embryonic epithelial			
rests in pituitary	572	<b>BACILLUS welchii</b> in biliary infec-	
— incidence of Dupuytren's contracture	540	tions	
— strangulated femoral hernia	181	Bacteriology of gall-bladder infections	619
— inguinal hernia	176	BAKER, S. L.: Pathological account of	
— umbilical hernia	186	parathyroid tumour associated	
Aged, acute appendicitis in	63	with fibrocystic disease	611
AINSWORTH-DAVIS, J. C.: Ureterocele:		BAND, DAVID, and HALL, I. SIMSON:	
some observations based on the		Post-operative massive collapse	
investigation and treatment of		of the lung: a clinical and ex-	
four cases	548	perimental study	
ALLEN-PRICE, E. D., and NEALE, A.		Barbitone derivatives in acute peritonitis	387
VICTOR: Chondrosarcoma of the		BARNETT, Sir Louis: Hydatid pneumo-	
fibula	47	thorax	
Amenorrhœa and obesity with hyper-		Bearded women, diabetes of	593
trichosis	563	Beds, plaster, technique of construction	563
Amputation, history of	171, 351	Benign osteogenic sarcoma	296
— the operation on Nelson in 1797	504	Bigelow (H. Jacob) on lithotripsy	330
Anæmia, effect on reactions of skin and		Biliary papilla, duodenal, carcinoma at,	
of tumours to radiation	481	pylephlebitis secondary to	336
Anaerobic infections of gall-bladder,		— passages, anaerobic infections of	619
incidence	619	BILLINGTON, WILLIAM: Excision of	
— peritoneum	638	subclavian aneurysm associated	
Anæsthesia, spinal, collapse of lung		with cervical rib	334
following	394	Billroth operations in chronic gastritis	45
Anæsthetics and pulmonary complica-		Bladder injured during herniotomy	179, 185
tions in hernia	191	— irritation in ureterocele	549



# INDEX

673

PAGE

Cholecystitis glandularis proliferans, terminology -	310
Cholelithiasis, anaerobic infection and -	619
Chondrosarcoma of fibula -	47
Civiale (Jean) on lithotrity -	1
Clavicle, fracture of, plaster splint for -	297
<b>Clinics at Home and Abroad:</b> -	
Dr. Murk Jansen at Leiden -	324
Clitoris in adrenal pseudo-hermaphroditism -	562
Collapse of lung, post-operative (see Lung) -	
Collar bone, fracture of, plaster splint for -	297
Colloid silver in pyelography -	622
Colon, resection of right half, preservation of ileocaecal sphincter in -	8
— sigmoid, torsion of appendix epiploica of -	508
COLT, G. H. : Three cases of torsion of an appendix epiploica of the sigmoid colon -	508
Congenital diaphragmatic hernia (see Diaphragmatic Hernia) -	
— eventration -	538
— lymphangiectatic fibrous hyper-trophy -	356
— nature of basic lesion of spondylolisthesis -	375
CONNELL, W. K. : The operative treatment of the elephantoid scrotum -	651
Constipation in strangulated femoral hernia -	182
— — hernia -	177
Contracture, Dupuytren's (see Dupuytren's Contracture) -	
CORBETT, C. H. : Anterior dislocation of the radius and its recurrence -	155
Cortex, adrenal, lesions of (see Adrenogenital Syndrome) -	
Costotransversectomy in Pott's paraplegia -	123
Cough reflex, abolition of, post-operative atelectasis and -	403, 404
Cyst formation in osteitis fibrosa -	258
— hydatid, of lung (see Hydatid Pneumothorax) -	
— mesenteric, in an infant six days old -	511
Cystic cholecystitis (see Cholecystitis Glandularis) -	
— pancreas and hypernephroma with papilliferous cystoma of petrous bone -	55
Cystoma of petrous bone with hypernephroma and cystic pancreas -	55
Cystoscopic appearances of ureterocele -	548, 551
— electrode in ureterocele -	550
DAVIE, T. B., and Ross, J. COSBIE : A case of suppurative pyelphlebitis secondary to carcinoma at the duodenal biliary papilla -	336
DAVIS, A. A. : The treatment of Dupuytren's contracture. A review of 31 cases, with an assessment of the comparative value of different methods of treatment -	539
Decompression in Pott's paraplegia -	122
DEVINE, H. B. : Exposure of the knee-joint -	306
'Diabetes of bearded women' -	559, 563
Diaphragm, post-operative collapse of lung and -	389, 393, 401
Diaphragmatic hernia -	527
— acquired -	535
— — eventration -	538
— — intra-abdominal pressure and -	536
— — non-traumatic -	535
— — traumatic -	538
— classification -	527
— congenital -	529
— causation -	527
— embryology -	531
— through the dome -	533
— — œsophageal wall -	529
— — pleuro-peritoneal hiatus -	533
— — thoracic stomach -	529
— types -	533
Diathermy, cystoscopic, in ureterocele -	529
Diet in etiology of osteomalacia -	550
Dislocation of hip, traumatic, complications -	276
— radius, anterior, and its recurrence -	601
Displacement of spine (see Spondylolisthesis) -	155
Dogs used in experimental production of atelectasis -	395
DUNNILL, T. P. : Carcinoma of the thyroid gland -	83
Duodenal biliary papilla, carcinoma at, pyelphlebitis secondary to -	336
Duodenitis phlegmonous -	362
Dupuytren's contracture -	539
— analysis of 31 cases -	540
— conclusions -	545
— etiology -	539
— radium and X-ray treatment -	545
— results of treatment -	541
— types of treatment -	540
EAR, middle, cancer of -	
Echinococcal cysts of lung (see Hydatid Pneumothorax) -	60
EMINOW, ALBERT, and MOTTAM, J. C. : On the effect of anaemia on the reactions of the skin and of tumours to radium exposure -	481
Elbow, dislocation of -	155
Electrode, cystoscopic, in ureterocele -	550
Elephantiasis angiomatosa -	360
Elephantoid serotum -	356
— criticism of operative methods -	651
— end-results of operation -	652
— establishment of new lymph pathway -	658

	PAGE		PAGE
Elephantoid serotum, hæmostasis in operation for - - -	652	Fistula, faecal, strangulated hernia and - - -	179, 182
— — — operative mortality - - -	658	— parotid, sialography in - - -	142, 144
— — — technique - - -	653	FITZGERALD, R. R.: Chronic follicular gastritis: with a report of nine cases - - -	25
— — — post-operative treatment - - -	657	FITZGERALD, M., and SWORN, B. R.: An analysis of 2126 cases of acute appendicitis - - -	410
— — — pre-operative treatment - - -	651, 653	Fluids contra-indicated in peritonitis - - -	638, 644
— — — summary - - -	658	Focal osteitis fibrosa - - -	268
Embolism, pulmonary, following hernia operations - - -	190	Fœtus, effect of increased intra-abdominal pressure on - - -	532
Embryo, effect of increased intra-abdominal pressure on - - -	532	Foley's insulated snare in ureterocoele - - -	550
Embryology of congenital diaphragmatic hernia - - -	527	Follicular gastritis, chronic ( <i>see</i> Gastritis, Chronic Follicular)	
— parathyroid glands - - -	645	FORBER, J. E.: Notes on statistical analysis of cases of cancer of breast treated by radium - - -	479
Embryonic epithelial rests in pituitary - - -	571	Fracture ( <i>see also</i> Splints)	
Enemata contra-indicated in peritonitis - - -	638	— of acetabulum with traumatic dislocation of hip - - -	601, 603, 605
Enteritis, phlegmonous, localized - - -	362	— carpal semilunar and Kienböck's disease ( <i>see</i> Kienböck's Disease)	
— — — analysis of recorded cases - - -	367	— femur with parathyroid tumour - - -	606
— — — cases from literature - - -	362	— — — with traumatic dislocation of hip - - -	603
— — — summary - - -	373	— tibial epiphysis - - -	78
— — — two case reports - - -	367	FRANKAU, CLAUDE: Strangulated hernia: a review of 1487 cases - - -	176
Enterostomy operations for strangulated hernia - - -	180	Friedrich's operation - - -	8
Epinephrectomy, unilateral, in cortical hyperplasia - - -	559	Frontal sinus, osteoma of, successful removal of - - -	158
Epiphysal growth at lower end of tibia, traumatic arrest of - - -	78		
Epithelial rests, embryonic, in pituitary - - -	571		
Eshelby (Thomas), operation on Lord Nelson by - - -	353		
Eventration - - -	537		
Extramucosal pyloromyotomy in chronic gastritis - - -	44		
<b>FACIES</b> in adreno-genital syndrome - - -	561, 563	<b>GALL-BLADDER</b> ( <i>see also</i> Cholecystitis)	
Fæcal fistula, strangulated hernia and - - -	179, 182	— anaerobic infections of, incidence - - -	619
Fasciotomy in Dupuytren's contracture - - -	541, 542	Gall-stones, anaerobic infection and - - -	619
Fat injections in Dupuytren's contracture - - -	542	Gas-gangrene serum in peritonitis - - -	638
Femoral hernia, strangulated - - -	181	Gastrectomy, partial, in chronic gastritis - - -	45
— thrombosis following hernia operations - - -	190	Gastric analysis in chronic follicular gastritis - - -	35
Femur, fracture of head, with traumatic dislocation of hip - - -	603	— ulcer, chronic, diagnosis from follicular gastritis - - -	44
— — — with parathyroid tumour - - -	606	Gastritis, chronic follicular - - -	25
Ferrier (David) on cerebral disease - - -	523	— — — case reports - - -	28
Fibrocystic disease of bones, generalized, case report - - -	192	— — — clinical and pathological summary of cases - - -	33
— — — with parathyroid tumour ( <i>see</i> Parathyroid Tumour)		— — — complications - - -	43
Fibrolysin injection in Dupuytren's contracture - - -	541, 542	— — — definition - - -	27
Fibromyoma of stomach weighing over 2 lb. - - -	339	— — — diagnosis - - -	43
Fibrosis, radium treatment of breast cancer and - - -	424, 427	— — — gross appearances - - -	36
Fibrous hypertrophy, congenital - - -	356	— — — historical - - -	25
Fibula, chondrosarcoma of - - -	47	— — — microscopical appearances - - -	37
Fingers in Dupuytren's contracture ( <i>see</i> Dupuytren's Contracture)		— — — prognosis - - -	44
Finsterer's signs of fracture of carpal semilunar - - -	579	— — — summary - - -	45
		— — — treatment - - -	44
		Gastro-photography controlled by post-mortem - - -	513
		Gastroscoy in chronic follicular gastritis - - -	36
		Gastrostaxis - - -	26
		Genitalia, external, in adreno-genital syndrome - - -	562

# INDEX

675

	PAGE		PAGE
GIBSON, A. : Regeneration of the inter-nal semilunar cartilage after operation -	302	Hernia, strangulated, post-operative complications -	190
Gigantism, local -	359	— — resections -	189
GIRDLESTONE, G. R. : The operative treatment of Pott's paraplegia -	121	— — a review of 1487 cases -	176
— — The pathology and treatment of tuberculous of knee-joint -	488	— — summary -	191
Godlee (Rickman), operation for cerebral tumour by -	524	— — umbilical -	186
Goitre, toxic -	87	— treatment in elephantoid serotum -	655
GORDON-TAYLOR, G., and WHITBY, LIONEL E. H. : The incidence of anaerobic infections in the gall-bladder -	619	Hessiau, use in constructing plaster splints -	293
— — WILES, PHILIP, and BAKER, S. L. : A case of parathyroid tumour associated with fibrocystic disease -	606	Heurteloup (Baron) on lithotrity -	1
GREENFIELD, J. G., et al. : The adrenogenital syndrome associated with cortical hyperplasia ; the results of unilateral adrenalectomy -	557	HILL, H. GARDINER, et al. : The adrenogenital syndrome associated with cortical hyperplasia ; the results of unilateral adrenalectomy -	557
Gum acacia, bronchoscopic introduction of, effect on respiratory tract and -	397	Hip, dislocation of, traumatic, complications -	601
Gunshot wounds of chest, atelectasis and -	394	Hirsutism and virilism, adrenal -	562
		HUME, J. BASIL : Diaphragmatic hernia -	527
HÆMATURIA in ureterocele -	549	Humerus, fracture of, plaster splint for -	298
— Hemorrhage complicating chronic follicular gastritis -	43	HUNTER, DONALD, and TURNBULL, HUBERT M. : Hyperparathyroidism : generalized osteitis fibrosa. With observations upon the bones, the parathyroid tumours, and normal parathyroid glands, Hunterian lecture, Jan. 21, 1931, by J. Basil Hume -	203
— from stomach -	26	Hydatid pneumothorax -	527
Hæmorrhagic erosions in chronic follicular gastritis -	41	— — case report -	593
Hæmostasis in operation for elephantoid serotum -	652	— — course of events in -	594
HALL, I. SIMSON, and BAND, DAVID : Post-operative massive collapse of the lung : a clinical and experimental study -	387	— — diagnosis -	596
Hand, congenital lymphangiectatic fibrous hypertrophy of -	356	— — rupture of cyst -	599
— in Dupuytren's contracture (see Dupuytren's Contracture) -	577	— — secondary development of cysts -	598
HARDING, H. E., MOUAT, T. B., and WILKIE, JOHN : Isolated fracture of the carpal semilunar and Kienböck's disease -	523	— — treatment -	599
Head, the first localized cerebral tumour -	557, 561	Hydrocele, treatment in elephantoid serotum -	655
Hernaphroditism, pseudo-, adrenal -	527	Hydronephrosis, intravenous pyelography in -	626, 627
Hernia, diaphragmatic (see Diaphragmatic Hernia) -	178, 183	Hyperealcæmia from over-dosage of parathyroid -	204, 260
— interstitial -	178	Hypernephroma and cystic pancreas with papilliferous cystoma of petrous bone -	55
— Littre's -	179	Hyperparathyroidism (see also Parathyroid Tumour) -	203
— Maydl's -	534	— animal experimentation with parathormone -	260
— para-oesophageal -	178, 183, 188	— biochemical aspect -	207
— reduction en masse -	176	— case reports -	213-51
— Richter's -	191	— cases from the literature -	207
— strangulated -	181	— clinical characteristics -	251
— anæsthetics and pulmonary emphysema -	176	— effects of parathyroid hormone -	260
— femoral -	176	— explanation of enlargement -	265
— inguinal -	176	— focal osteitis fibrosa and -	268
		— generalized osteitis fibrosa -	205
		— — pathogenesis -	254
		— methods of study -	212
		— operative technique -	253
		— osteomalacia and (see also Osteomalacia) -	256, 272
		— Paget's osteitis deformans and -	268
		— relation of parathyroid tumour to changes in bone -	206
		— surgical treatment -	285
		— — case reports -	289



	PAGE		PAGE
Hyperparathyroidism, X-ray examination of bone changes - - -	213	KEYNES, GEOFFREY: The radium treatment of carcinoma of the breast - - -	415
Hyperplasia, adrenal cortical ( <i>see</i> Adreno-genital Syndrome)		Kidney ( <i>see</i> Pyclography; Renal)	
Hypertrichosis with obesity and amenorrhoea - - -	563	Kienböck's disease - - -	577
Hypertrophy, congenital lymphangiectatic fibrous - - -	356	— case reports - - -	578
		— clinical signs and symptoms - - -	588
		— discussion - - -	587
		— etiology - - -	588
		— histological findings - - -	589
		— prognosis and treatment - - -	590
		— summary - - -	592
		— workmen's compensation in - - -	591
		— X-ray appearances - - -	589
I LEOCAECAL sphincter, preservation of, in resection of right half of colon - - -	8	KING, E. S. J.: An example of benign osteogenic sarcoma - - -	330
— — spasm and incompetence of - - -	10	— — and MACCALLUM, P.: Cholecystitis glandularis proliferans (cystica) - - -	310
— — structure, movements, and function of - - -	9	Knee-joint, benign osteogenic sarcoma of - - -	330
— — transplantation of, case reports - - -	17	— exposure of, new technique - - -	306
— — — discussion - - -	15	— regeneration of semilunar cartilage after operation - - -	302
— — — technique - - -	10	— tuberculosis of - - -	488
Illuminating retractor for intracranial surgery - - -	69	— — ages for operating on - - -	490, 500
Infant, six-day-old, mesenteric cyst in Infraclavicular lymphatics, radium treatment of, in breast cancer - - -	511	— — diagnosis - - -	493
Inguinal hernia, strangulated - - -	176	— — diagnostic arthrotomy - - -	495
Insulated snare in ureterocoele - - -	550	— — 'extra-articular' group - - -	489
Intercostal spaces, radium treatment of, in breast cancer - - -	422, 438	— — 'focal articular' group - - -	490
Interstitial hernia - - -	178	— — methods of immobilization - - -	498, 502
Intervertebral discs in spondylolisthesis	382	— — 'non-focal' group - - -	491
Intestinal obstruction following herniotomy - - -	185	— — pathology - - -	488
— — in general peritonitis - - -	635	— — site of infection - - -	489
Intestine, small, carcinoma of, two cases	149	— — summary - - -	506
Intra-abdominal pressure, increase in, effect on embryo - - -	532	— — synovial - - -	491
— — rôle in producing hernia - - -	536	— — treatment, general and local conservative - - -	498
Intracranial tumours (pituitary), radon-seed treatment - - -	68, 659	— — — operative - - -	499
Intraperitoneal abscess - - -	638	— — — amputation - - -	504
Intravenous pyclography ( <i>see</i> Pyclography)		— — — arthroplasty - - -	505
Iodides of potassium and sodium in pyclography - - -	623	— — — excision, fusion, and arthrodesis - - -	499
Iodine in Dupuytren's contracture - - -	542	— — — indications for excision and contra-indications - - -	500
IRWIN, C. GORDON, and McDONALD, STUART: Two cases of localized phlegmonous enteritis - - -	362	— — — results of excision - - -	502
Isolated fracture of carpal semilunar and Kienböck's disease ( <i>see</i> Kienböck's Disease)		— — — sinus formation and - - -	505
		— — — synovectomy - - -	501
		— — — technical points - - -	500
		— — — value of clinical diagnosis - - -	495
J ANSEN'S (Murk) clinic at Leiden	324	L ACTATION after radium treatment of breast cancer - - -	446
Jaundice in carcinoma at duodenal biliary papilla - - -	336	Laminae, X-ray appearance of, in spondylolisthesis - - -	382
JEFFERSON, GEOFFREY, and WHITEHEAD, RAYMOND: Papilliferous cystoma of the petrous bone associated with hypernephroma and cystic pancreas - - -	55	Laminectomy and graft operation in Pott's paraplegia, technique - - -	137
Jejunum, condition of, in phlegmonous enteritis - - -	368, 371	Laparotomy, experimental investigation of effect on respiratory tract - - -	395, 397
Jensen rat sarcoma, effect of anaemia on radiation of - - -	484	Lateral aberrant thyroids - - -	92
		Leg, injuries to, plaster splint for - - -	300
		Leiden, Murk Jansen's clinic at - - -	324
		LEWIS, J.: A clinical study of acute appendicitis in old age - - -	63



	PAGE		PAGE
Old age, acute appendicitis in - - -	63	Perigastric adhesions complicating	
Omentum, condition in strangulated		chronic follicular gastritis - - -	43
hernia - - - - -	179, 184	Peristalsis, visible, in strangulated	
O'SHAUGHNESSY, LAWRENCE: A tumour		femoral hernia - - - - -	182
of the carotid body - - - - -	133	Peritoneal cavity in phlegmonous en-	
Osteitis deformans, Paget's - - - -	266	teritis - - - - -	367
— fibrosa, generalized ( <i>see</i> Hyperpara-		Peritonitis, acute general, atropine in	
thyroidism)		639, 643	
Osteoclastomata in osteitis fibrosa		— — — case reports - - - - -	640
223, 242, 259		— — — clinical picture - - - - -	634
Osteogenic sarcoma, benign - - - -	330	— — — critique of organic theory of	
Osteoma of frontal sinus, successful re-		obstruction in - - - - -	635
moval of - - - - -	158	— — — direct observations of abdo-	
Osteomalacia - - - - -	256, 272	men in - - - - -	635
— case report - - - - -	277	— — — intestinal obstruction in - - -	635
— clinical features - - - - -	274	— — — isolation of bowel from sym-	
— economic factors causing - - - -	276	pathetic stimuli - - - - -	639
— pathogenesis - - - - -	275	— — — morphia in - - - - -	638, 643
— treatment - - - - -	276	— — — physiology - - - - -	636
Otosclerosis with hyperparathyroidism	242	— — — sympathetic in - - - - -	634
Oxyphil cells of parathyroid glands -	220	— — — treatment - - - - -	637
		— — — — of hyperactivity of sym-	
PAGET'S osteitis deformans - - - -	266	pathetic - - - - -	638
Pain in strangulated femoral		— with appendicitis, analysis of 231	
hernia - - - - -	182	cases - - - - -	410
— — inguinal hernia - - - - -	177	Per-urethral treatment of ureterocele	550
Pancreas, cystic, and hypernephroma,		Petrous bone, papilliferous cystoma of,	
with papilliferous cystoma of		with hypernephroma and cystic	
petrous bone - - - - -	55	pancreas - - - - -	55
Papilla, duodenal biliary, carcinoma at,		Phlegmonous enteritis, localized ( <i>see</i>	
pyelephlebitis secondary to - - -	336	Enteritis)	
Papilliferous adenocarcinoma of thyroid		Phosphorus and calcium blood-count in	
gland ( <i>see</i> Thyroid Gland)		parathyroid tumour 606, 608, 616	
— cystoma of petrous bone with hyper-		— excretion, effect of parathormone on	
nephroma and cystic pancreas - -	55	203, 260	
Parathormone, effects on calcium and		Phrenicotomy, post-operative collapse	
phosphorus excretion - 203, 260		of lung and - - - - -	389, 401
— — of over-dosage - - - - -	204, 260	Pin-hole orifice of ureter - - - - -	548
— for tetany after removal of parathy-		Pituitary gland, embryonic epithelial	
roid tumour - - - - -	608, 617	rests in - - - - -	571
Parathyroid glands, embryology - - -	645	— tumours, radon-seed treatment 68, 659	
— — hyperfunction of ( <i>see</i> Hyperpara-		Pituitrin in acute appendicitis - - -	644
thyroidism)		Plaster spica in tuberculosis of knee 498, 502	
— — structure - - - - -	220	— splints ( <i>see</i> Splints)	
— rest, cancer of neck arising in - -	645	PLATT, HARRY: On some complications	
— tumour, fibrocystic disease with -	606	of traumatic dislocation of the	
— — — calcium and phosphorus		hip-joint - - - - -	601
blood-count in 606, 608, 616		Pleuro-pericardial ridges, embryology	528
— — — — clinical report - - - - -	606	Pleuro-peritoneal hiatus, hernia through	529
— — — — histology of bones - - -	611	Pneumothorax, hydatid ( <i>see</i> Hydatid	
— — — — pathology of tumour - -	615	Pneumothorax)	
— — tetany after removal - - - -	608, 617	Polya operation in chronic gastritis -	45
Para-oesophageal hernia - - - - -	534	Post-operative massive collapse of lung	
Parotid ducts, X-ray examination of -	142	( <i>see</i> Lung)	
Parotitis, subacute, sialography in		Posture in production of post-operative	
142, 144		atelectasis - - - - -	394
PAYNE, REGINALD T.: Sialography;		Potassium iodide in pycelography -	623
its technique and applications -	142	Pott's fracture, plaster splint for -	300
Payr's extramucosal pyloromyotomy in		— paraplegia - - - - -	121
chronic gastritis - - - - -	44	— — argument for operative treatment	121
Pelvis, rotation of, in spondylolisthesis	381	— — case reports - - - - -	125
Peri-arterial sympathectomy, time dur-		— — cototransversectomy in - - -	123
ing which vascular responses		— — post-operative treatment - - -	141
persist after - - - - -	52	— — technique of combined laminec-	
		tomomy and graft operation - - -	137

	PAGE		PAGE
POWER, Sir D'ARCY : Amputation : the operation on Nelson in 1797 - - -	171, 351	Rats in experiments to show effect of anaemia on radiation - - -	481
— — The first localized cerebral tumour - - -	523	RAWLING, L. BATHE : A contribution to the surgery of the pituitary region. An account of four cases of 'pituitary tumour' treated by radon seeds - - -	68
— — Lithotripsy : the case of the Emperor Napoleon III - - -	1	— — Note on a case of pituitary tumour treated by radon seeds - - -	659
Precocity, adrenal - - -	558	Recklinghausen's (von) disease ( <i>see Hyperparathyroidism</i> ) - - -	179
Pregnancy after cancer of breast treated by radium - - -	446	Reduction en masse - - -	302
Pre-spondylolisthesis - - -	376	Regeneration of internal semilunar cartilage after operation - - -	213, 233
PRICE, L. WOODHOUSE, and MOWAT, G. T. : A case of rapidly-growing carcinoma of the neck, arising in a parathyroid rest - - -	645	Renal calculi with hyperparathyroidism - - -	628, 630
Pseudo-hermaphroditism, adrenal - - -	557, 561	— — intravenous pyelography in - - -	549
Psychic effects of follicular gastritis - - -	44	— pain in ureterocele - - -	403, 404
Pubic hair in adreno-genital syndrome - - -	561, 563	Respiratory movement, limitation of, post-operative atelectasis and - - -	571
Pulmonary ( <i>see Lung</i> ) - - -	-	Rests, embryonic epithelial, in pituitary surgery - - -	69
Pyelognost, use in pyelography - - -	623	Reviews and Notices of Books (Titles) :-	-
Pyelography, intravenous - - -	622	<i>Abdominal pain</i> (J. Morley) - - -	517
— — case reports - - -	624	<i>Anatomy</i> (G. W. Corner) - - -	159
— — conclusions - - -	632	<i>Annals of roentgenology</i> (Vol. xii) : <i>The chest in children</i> (E. G. Stoloff)	165
— — historical account - - -	622	<i>Der appendicitische Anfall</i> (L. Aschoff) - - -	349
— — recent media for - - -	624	<i>Die Avertinmarkose in der Chirurgie</i> (D. W. Anschütz, K. Specht, and F. R. Tiemann) - - -	348
Pylephlebitis secondary to carcinoma at duodenal biliary papilla - - -	336	<i>The beginnings : Egypt and Syria</i> (W. R. Dawson) - - -	159
Pylorectomy in chronic gastritis - - -	45	<i>Le cancer</i> (G. Roussy) - - -	166
Pyloric stenosis complicating chronic follicular gastritis - - -	43	<i>Cancer and race</i> (M. Sorsby) - - -	166
— syndrome 'of Soupault' - - -	26	<i>The chest in children</i> (E. G. Stoloff)	165
Pyloromyotomy, extramucosal, in chronic gastritis - - -	44	<i>Chirurgia del dolore</i> (I. Scalzone) - - -	346
		<i>Die Chirurgie</i> , Fasc. 30 (ed. M. Kirschner and O. Nordmann) - - -	345
		<i>Chirurgie de l'ulcère gastrique et duodénal</i> (N. Hortolmei and VI E. Butureanu) - - -	518
		<i>Clinical examination and surgical diagnosis</i> (F. Lejars, trans. Helen C. Scott) - - -	666
		<i>Clinical observations on the surgical pathology of bone</i> (D. M. Greig) - - -	518
		<i>Clio Medica Series</i> (ed. E. B. Krumblaar) - - -	159
		<i>Collected papers 1924-1929</i> (E. Beer)	342
		<i>Crippled children : their treatment and orthopedic nursing</i> (E. D. McBride) - - -	344
		<i>Deep X-ray therapy in malignant disease</i> (W. M. Levitt) - - -	164
		<i>Diseases of the thyroid gland</i> (C. A. Joll) - - -	661
		<i>Diseases of the tongue</i> (W. G. Spence and S. Cade) - - -	163
		<i>Die Elektrochirurgie</i> (F. Keysser) - - -	668
		<i>Emergency surgery</i> , Vol. ii (H. Bailey) - - -	664

	PAGE
Reviews and Notices of Books, <i>contd.</i> —	
<i>On faith and science in surgery</i> (Sir J. Bland-Sutton) - - -	516
<i>Fractures and their complications</i> (G. E. Wilson) - - -	165
<i>Fractures of the jaw</i> (R. H. Ivy and L. Curtis) - - -	667
<i>Gonococcal infection in the male</i> (A. L. Woolbarst) - - -	520
<i>Grundzüge der Neurochirurgie</i> (W. Lehmann) - - -	519
<i>Guy's Hospital reports</i> , Vol. lxxxix (ed. A. F. Hurst) - - -	168
<i>Indikation und Technik des künstlichen Pneumothorax</i> (H. Mayrhofer) - - -	668
<i>Injuries to joints</i> (Sir R. Jones) - -	346
<i>Injuries and sport</i> (C. B. Heald) - -	520
<i>Internal medicine</i> (Sir H. Rolleston) -	159
<i>The intervertebral discs</i> (O. A. Beadle) -	667
<i>Der künstliche Pneumothorax</i> (H. Alexander) - - -	668
<i>The management of abdominal operations</i> (R. H. Maingot) - -	517
<i>Medicine in the British Isles</i> (Sir D'Arcy Power) - - -	159
<i>Medizinische Praxis Sammlung für ärztliche Fortbildung</i> , Vol. vii (W. Lehmann) - - -	519
<i>The metabolism of tumours</i> (O. Warburg, trans. F. Dickens) - -	168
<i>Minor surgery and bandaging</i> (G. Williams) - - -	343
<i>Modern surgery</i> (C. J. Da Costa and B. Lipshutz) - - -	516
<i>Nasenplastik und sonstige Gesichtsplastik, etc.</i> (J. Joseph) - - -	341
<i>Operative Chirurgie der Knochenbrüche</i> (F. König) - - -	345
<i>Operative surgery</i> (M. Kirschner, trans. I. S. Ravdin) - - -	517
<i>Physiologie pathologique chirurgicale</i> (R. Leriche and A. Policard) -	167
<i>Physiology</i> (J. F. Fulton) - - -	159
<i>Practical methods in the diagnosis and treatment of venereal diseases</i> (D. Lees) - - -	347
<i>La pratique chirurgicale illustrée</i> , Fasc. xvii (V. Pauchet) - - -	664
<i>Pye's surgical handcraft</i> (ed. H. W. Carson) - - -	663
<i>Radium therapy: principles and practice</i> (G. E. Birkett) - - -	165
<i>La sacro-coxalgie et son traitement</i> (P. Ingelsans) - - -	519
<i>Sir D'Arcy Power: selected writings 1877-1930</i> - - -	160
<i>Surgery: its principles and practice</i> (A. P. C. Ashhurst) - - -	343
<i>Surgical diagnosis</i> (ed. E. A. Graham) - - -	168
<i>Surgical pathology of the genitourinary organs</i> (A. E. Hertzler) -	666
<i>Surgical pathology of the skin, fascia, etc.</i> (A. E. Hertzler) - - -	519

	PAGE
Reviews and Notices of Books, <i>contd.</i> —	
<i>A textbook of surgery</i> (J. Homans) -	515
<i>A text-book of the surgical dyspepsias</i> (A. J. Walton) - - -	162
<i>A textbook on surgical pathology</i> (C. F. W. Illingworth and B. M. Dick) - - -	665
<i>Thomson and Miles' manual of surgery</i> (A. Miles and D. P. D. Wilkie) -	344, 663
<i>Le traitement médico-chirurgical de l'occlusion intestinale aiguë et subaiguë</i> (P. Moiroud) - - -	349
<i>Verlauf der wichtigsten Knochen- im Röntgen</i> - - -	346

### Authors:—

Alexander, H.: <i>Der künstliche Pneumothorax</i> - - -	668
Anschütz, D. W., et al.: <i>Die Aver-tinarkose in der Chirurgie</i> - - -	348
Aschoff, L.: <i>Der appendicitische Anfall</i> - - -	349
Ashhurst, A. P. C.: <i>Surgery: its principles and practice</i> - - -	343
Bailey, H.: <i>Emergency surgery</i> , Vol. ii - - -	664
Beadle, O. A.: <i>The intervertebral discs</i> - - -	667
Beer, E.: <i>Collected papers 1904-1929</i> - - -	342
Birkett, G. E.: <i>Radium therapy principles and practice</i> - - -	165
Bland-Sutton, Sir J.: <i>On faith and science in surgery</i> - - -	516
Butureanu, Vl., and Hortolmei, N.: <i>Chirurgie de l'ulcère gastrique et duodénal</i> - - -	518
Cade, S., and Spence, W. G.: <i>Diseases of the tongue</i> - - -	163
Carson, H. W.: <i>Pye's surgical handcraft</i> (ed.) - - -	663
Cass, J. T.: <i>Annals of roentgenology</i> , Vol. xii (ed.) - - -	165
Cornor, G. W.: <i>Anatomy</i> - - -	159
Curtis, L., Ivy, R. H.: <i>Fractures of the jaws</i> - - -	667
Da Costa, C. J., and Lipshutz, B.: <i>Modern surgery</i> - - -	516
Dawson, W. R.: <i>The beginnings: Egypt and Syria</i> - - -	159
Dick, B.M., and Illingworth, C. F. W.: <i>A textbook on surgical pathology</i> - - -	665
Dickens, F.: <i>The metabolism of tumours</i> (trans.) - - -	168
Fromme, A., et al.: <i>Medizinische Praxis Sammlung für ärztliche Fortbildung</i> , Vol. vii (ed.) - -	519
Fulton, J. F.: <i>Physiology</i> - - -	159
Graham, E. A.: <i>Surgical diagnosis</i> (ed.) - - -	168
Greig, D. M.: <i>Clinical observations on the surgical pathology of bone</i> -	518

# INDEX

681

## Reviews and Notices of Books, contd.—

Grote, L. R., et al. : <i>Medizinische Praxis Sammlung für ärztliche Fortbildung</i> , Vol. vii (ed.)	591
Herald, C. B. : <i>Injuries and sport of the genito-urinary organs</i>	520
Hertzler, A. E. : <i>Surgical pathology of the genito-urinary organs</i>	606
— — <i>Surgical pathology of the skin, fasciae, etc.</i>	519
Hoffmann, V. : <i>Verlauf der wichtigsten Knochen- und Gelenkerkrankungen im Röntgenbilde</i>	346
Homans, J. : <i>A text-book of surgery</i>	515
Hortolmei, N., and Butureau, V. : <i>Chirurgie de l'ulcère gastrique et duodénal</i>	518
Hurst, A. F. : <i>Guy's Hospital reports</i> , Vol. lxxxvi (ed.)	168
Illingworth, C. F. W., and Dick, B. M. : <i>A textbook on surgical pathology</i>	665
Ingelsans, P. : <i>La sacro-coxalgie et son traitement</i>	519
Ivy, R. H., and Curtis, L. : <i>Fractures of the jaw</i>	667
Joll, C. A. : <i>Diseases of the thyroid gland</i>	661
Jones, Sir R. : <i>Injuries to joints</i>	346
Joseph, J. : <i>Nasenplastik und sonstige Gesichtsplastik, etc.</i>	341
Keysser, F. : <i>Die Elektrochirurgie</i>	668
Kirschner, M. : <i>Operative surgery</i> (trans. I. S. Ravdin)	517
— — and Nordmann, O. : <i>Die Chirurgie</i> , Fasc. 30 (ed.)	345
König, F. : <i>Operative Chirurgie der Knochenbrüche</i>	345
Krumblhaar, E. B. : <i>Clio Medica Series</i> (ed.)	159
Lees, D. : <i>Practical methods in the diagnosis and treatment of venereal diseases</i>	347
Lehmann, W. : <i>Grundzüge der Neurochirurgie</i>	519
Lejars, F. : <i>Clinical examination and surgical diagnosis</i> (trans. Helen C. Scott)	666
Leriche, R., and Policard, A. : <i>Physiologie pathologique chirurgicale</i>	167
Levitt, W. M. : <i>Deep X-ray therapy in malignant disease</i>	164
Lipshutz, B., and DaCosta, C. J. : <i>Modern surgery</i>	516
McBride, E. D. : <i>Crippled children : their treatment and orthopedic nursing</i>	344
Maingot, R. H. : <i>The management of abdominal operations</i>	517
Mayrhofer, H. : <i>Indikation und Technik des künstlichen Pneumothorax</i>	668
Miles, A., and Wilkie, D. P. D. : <i>Thomson and Miles' manual of surgery</i>	344, 663

PAGE

## Reviews and Notices of Books, contd.—

PAGE

Moiroud, P. : <i>Le traitement médical chirurgical de l'occlusion intestinale aiguë et subaiguë</i>	349
Morley, J. : <i>Abdominal pain</i>	517
Nordmann, O., and Kirschner, M. : <i>Die Chirurgie</i> , Fasc. 30 (ed.)	345
Pauehet, V. : <i>La pratique chirurgicale illustrée</i> , Fasc. xvii	664
Policard, A., and Leriche, R. : <i>Physiologie pathologique chirurgicale</i>	167
Power, Sir D'Arey : <i>Medicine in the British Isles</i>	159
— — <i>selected writings, 1877-1930</i> (trans.)	160
Ravdin, I. S. : <i>Operative surgery</i> (trans.)	517
Rolleston, Sir H. : <i>Internal medicine</i>	159
Roussy, G. : <i>Le cancer</i>	166
Sealone, I. : <i>Chirurgia del dolore</i>	346
Scott, Helen C. : <i>Clinical examination and surgical diagnosis</i> (trans.)	666
Sorsby, M. : <i>Cancer and race</i>	166
Specht, K., et al. : <i>Die Avertinnarkose in der Chirurgie</i>	348
Spencer, W. G., and Cade, S. : <i>Diseases of the tongue</i>	163
Stoloff, E. G. : <i>The chest in children</i>	165
Tiemann, Fr., et al. : <i>Die Avertinnarkose in der Chirurgie</i>	348
Walton, A. J. : <i>A text-book of the surgical dyspepsias</i>	162
Warburg, O. : <i>The metabolism of tumours</i> (trans. F. Dickens)	168
Warnekros, K. : <i>Medizinische Praxis Sammlung für ärztliche Fortbildung</i> , Vol. vii (ed.)	519
Wilkie, D. P. D., and Miles, A. : <i>Thomson and Miles' manual of surgery</i>	344, 663
Williams, G. : <i>Minor surgery and bandaging</i>	343
Wilson, G. E. : <i>Fractures and their complications</i>	165
Woolbarst, A. L. : <i>Gonococcal infection in the male</i>	520
Rib, cervical, subclavian aneurysm associated with	334
Richter's hernia	178, 183, 188
Rickets, etiology	274
ROBB, JOHN J. : <i>The sympathetic in acute general peritonitis. A clinical study with observations on treatment</i>	634
ROGERS, LAMBERT : <i>The time factor during which vascular responses persist following periarterial sympathectomy</i>	52
ROSS, J. COSBIE, and DAVIE, T. B. : <i>A case of suppurative pyelophlebitis secondary to carcinoma at the duodenal biliary papilla</i>	336
Rotato-scoliosis	378

	PAGE		PAGE
SACRUM, rôle in etiology of spondylolisthesis - - - - -	376, 384	Spina bifida, spondylolisthesis and - - - - -	378
SAINT, JAMES H., and WILLAN, R. J. : Further studies in intravenous pyelography - - - - -	622	Spinal anæsthesia, collapse of lung fol- lowing - - - - -	394
Salivary glands, X-ray examination of ducts of - - - - -	142	Spine, caries of ( <i>see</i> Pott's Paraplegia) — displacement of ( <i>see</i> Spondylolis- thesis)	
Sarcoma, benign osteogenic - - - - -	330	— injuries of, plaster beds for - - - - -	296
— of fibula - - - - -	47	Splint, caliper, in tuberculosis of knee 498, 502	
— Jensen rat, the effect of anæmia on radiation of - - - - -	484	Splints, plaster - - - - -	292
Sciatic nerve involved in traumatic dislocation of hip - - - - -	602, 603	— — beds for spinal cases - - - - -	296
Seirrhus of thyroid gland ( <i>see</i> Thyroid Gland)		— — for fractured clavicle - - - - -	297
Scoliosis with spondylolisthesis - - - - -	378	— — — humerus - - - - -	298
Serim, use in constructing plaster splints	293	— — — lower limbs - - - - -	300
Serotum, elephantoid ( <i>see</i> Elephantoid Scrotum)		— — materials used - - - - -	293
Selectan-neutral in pyelography - - - - -	624	— — summary - - - - -	301
Semilunar, carpal, fracture of, Kien- böck's disease and ( <i>see also</i> Kienböck's Disease) - - - - -	377	— — technique of construction - - - - -	293
— cartilage, internal, regeneration after operation - - - - -	302	— — for wrist injuries - - - - -	299
Septum transversum, embryology - - - - -	528	Spondylolisthesis - - - - -	374
Serum, anti-gas-gangrene, in treatment of peritonitis - - - - -	638	— clinical features - - - - -	380
— calcium, effect of parathormone on 203, 260		— congenital nature of basic lesion - - - - -	375
— — and phosphorus in parathyroid tumour - - - - -	606, 608, 616	— factors limiting displacement - - - - -	380
Sex incidence of Dupuytren's contrac- ture - - - - -	540	— literature of - - - - -	375
Shock in strangulated inguinal hernia - - - - -	177	— rôle of trauma in development of - - - - -	375
Sialography - - - - -	142	— rotato-scoliosis with - - - - -	378
— case reports - - - - -	144	— spina bifida and - - - - -	378
— indications - - - - -	142	— summary - - - - -	384
— summary - - - - -	147	— treatment - - - - -	384
— technique - - - - -	142	— types - - - - -	374
Sigmoid colon, torsion of appendix epi- ploica of - - - - -	508	— X-ray appearances - - - - -	381
Silver preparations in pyelography - - - - -	622	Spondylolysis - - - - -	374, 377
Sinus formation in tuberculosis of knee - - - - -	505	Stenosis, pyloric, complicating chronic follicular gastritis - - - - -	43
— frontal, successful removal of - - - - -	158	STEWART, H. HAMILTON : The pathology and treatment of tumours of the carotid body - - - - -	114
Skin nodules following radium treat- ment of breast cancer - - - - -	451	Stomach ( <i>see also</i> Gastric ; Gastritis) — cancer of, diagnosis from follicular gastritis - - - - -	44
— œdema of, following radium treat- ment of breast cancer - - - - -	424	— — gastro-photograph controlled by post-mortem - - - - -	513
— reactions to radiation, effect of anæmia on - - - - -	481	— fibromyoma of, weighing over 2 lb. - - - - -	339
Skin-flaps in operation on elephantoid scrotum - - - - -	652, 654	— hæmorrhage from - - - - -	26
Skin-grafting in Dupuytren's contrac- ture - - - - -	543	— thoracic, hernia through - - - - -	533
Skull, the first localized cerebral tumour	523	Strangulated hernia ( <i>see</i> Hernia)	
Snare, insulated, in ureterocele - - - - -	550	Subclavian aneurysm associated with cervical rib - - - - -	334
Sodium bromide in pyelography - - - - -	623	Subcutaneous tissue transplantation in Dupuytren's contracture - - - - -	544
— iodide in pyelography - - - - -	623	Subpectoral lymphatics, radium treat- ment of, in breast cancer - - - - -	420
Soupaault's 'pyloric syndrome' - - - - -	26	Supraclavicular lymphatics, radium treatment of, in breast cancer 421, 438	
Spasm and incompetence of ileocecal sphincter - - - - -	10	Suprapubic treatment of ureterocele - - - - -	550
Sphincter, ileocecal ( <i>see</i> Ileocecal Sphincter)		Suprarenal cortex, lesions of ( <i>see</i> Adreno- genital Syndrome)	
		<b>Surgical Clinics at Home and Abroad :—</b>	
		Dr. Murk Jansen and his clinic at Leiden - - - - -	324
		SUSMAN, WILLIAM : Embryonic epithe- lial rests in the pituitary - - - - -	571
		SWORN, B. R., and FITZGIBBON, M. : An analysis of 2126 cases of acute appendicitis - - - - -	410





	PAGE		PAGE
WHIGHAM, J. R. M.: Gastro-photo- graphy controlled by post- mortem examination - - -	513	Woodall (John) on amputation - -	171
WHITNY, LIONEL E. H., and GORDON- TAYLOR, G.: The incidence of anaerobic infections in the gall- bladder - - -	619	Wounds of chest, massive collapse of lung and - - -	394
WHITEHEAD, RAYMOND, and JEFFERSON, GEOFFREY: Papilliferous cyst- oma of the petrous bone associ- ated with hypernephroma and cystic pancreas - - -	55	Wrist, injuries to, plaster splint for -	299
WILES, PHILIP, GORDON-TAYLOR G., and BAKER, S. L.: A case of parathyroid tumour associated with fibrocystic disease - - -	606	— lesions of ( <i>see</i> Kienböck's Disease)	
WILKIE, JOHN, MOUAT, T. B., and HARDING, H. E.: Isolated frac- ture of the carpal semilunar and Kienböck's disease - - -	577	X-RAY appearances of generalized fibrocystic disease of the bones -	193
WILLAN, R. J., and SAINT, JAMES H.: Further studies in intravenous pyelography - - -	622	— — Kienböck's disease - - -	589
WILLIAMS, O. HERBERT, WILLIAMS, W. ROBERT, and MOLE, R. HOWARD: Two cases of carcinoma of the small intestine - - -	149	— — post-operative massive collapse of lung - - -	388
		— — spondylolisthesis - - -	381
		— diagnosis of tuberculosis of knee -	493
		— — urinary tract affections ( <i>see</i> Pyc- elography)	
		— examination after transplantation of ileocaecal sphincter - - -	17
		— — in chronic follicular gastritis -	36
		— — of ducts of salivary glands -	142
		— — generalized osteitis fibrosa -	213
		— therapy in cancer of thyroid gland -	107
		— — Dupuytren's contracture - -	545
		YONGE (James) on amputations -	172

# ATLAS OF PATHOLOGICAL ANATOMY

ISSUED UNDER THE DIRECTION OF THE EDITORIAL COMMITTEE OF  
*The British Journal of Surgery.*

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FASCICULUS VII.

DISEASES OF THE THYROID GLAND.  
DISEASES OF THE MOUTH, PHARYNX,  
AND ŒSOPHAGUS.

Compiled by E. K. MARTIN, M.S., F.R.C.S.

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# CONTENTS

	PAGE
EXOPHTHALMIC GOITRE - - - - -	97
The thyroid and thymus glands together with the larynx and a part of the trachea <i>p.</i> 98	
Microscopical section <i>p.</i> 99	
CHRONIC THYROIDITIS - - - - -	100
CHRONIC THYROIDITIS (RIEDEL'S DISEASE) - - - - -	101
The cut surface of part of a thyroid gland drawn from behind ( <i>coloured</i> ) <i>p.</i> 102	
Microscopical section <i>p.</i> 103	
CHRONIC THYROIDITIS (RIEDEL'S DISEASE) - - - - -	104
A thyroid gland, part of which has been removed ( <i>coloured</i> ) <i>p.</i> 105	
MALIGNANT DISEASE OF THE THYROID GLAND - - - - -	106
CARCINOMA OF THYROID - - - - -	107
The right lobe of a thyroid gland in section ( <i>coloured</i> ) <i>p.</i> 107	
Microscopical section of primary growth <i>p.</i> 108	
Microscopical section of secondary growth in lung <i>p.</i> 109	
CARCINOMA OF THYROID (LOCAL RECURRENCE) - - - - -	110
The anterior half of a coronal section through the neck <i>p.</i> 111	
CARCINOMA OF THYROID (SECONDARY GROWTH IN BONE) - - - - -	112
Portions of two lumbar vertebræ in sagittal section ( <i>coloured</i> ) <i>p.</i> 112	
Microscopical section <i>p.</i> 113	
CARCINOMA OF THYROID - - - - -	114
A larynx and trachea with a portion of the thyroid gland drawn from the front ( <i>coloured</i> ) <i>p.</i> 114	
CARCINOMA OF THYROID (SECONDARY GROWTH IN SKULL) - - - - -	115
Part of the left side of a skull ( <i>coloured</i> ) <i>p.</i> 115	
PROLIFERATING ADENOMA OF THYROID (TYPE LANGHANS) - - - - -	116
The right lobe of a thyroid gland ( <i>coloured</i> ) <i>p.</i> 117	
CARCINOMA OF THYROID - - - - -	118
One half of a vertical section through the right lobe of the thyroid gland and the trachea ( <i>coloured</i> ) <i>p.</i> 119	
Microscopical section <i>p.</i> 120	
INFLAMMATION, LEUCOPLAKIA, AND ULCERATION OF THE TONGUE - - - - -	121
ACUTE PARENCHYMATOUS GLOSSITIS (ABSCESS OF TONGUE) - - - - -	122
A tongue with the larynx and pharynx. The pharynx has been opened from the back ( <i>coloured</i> ) <i>p.</i> 123	
TUBERCULOSIS AND SYPHILIS OF THE TONGUE - - - - -	124
TUBERCULOUS ULCER OF TONGUE - - - - -	125
A tongue drawn from the dorsum ( <i>coloured</i> ) <i>p.</i> 125	
SIMPLE TUMOURS OF THE TONGUE - - - - -	126
HEMANGIOMA OF TONGUE - - - - -	127
The right half of a tongue <i>p.</i> 127	

	PAGE
ANGIOMA OF TONGUE - - - - -	128
Tongue removed by operation. The substance is formed by a spongy mass of dilated blood-channels p. 128	
ENDOTHELIOMA OF TONGUE - - - - -	129
The right half of a tongue p. 129	
PERITHELIOMA OF TONGUE - - - - -	130
Part of a tongue divided by longitudinal section p. 130	
PAPILLOMA OF TONGUE - - - - -	131
The right half of a tongue p. 131	
THYROGLOSSAL CYST OF TONGUE - - - - -	132
The tongue of a child divided by sagittal section p. 132	
MALIGNANT TUMOURS OF THE TONGUE AND MOUTH - - - - -	133
CARCINOMA OF TONGUE - - - - -	134
One half of a tongue which has been divided by sagittal section (coloured) p. 134	
PAPILLOMA AND CARCINOMA OF TONGUE - - - - -	135
A tongue drawn from the dorsal aspect (coloured) p. 135	
CARCINOMA OF TONGUE - - - - -	136
A horizontal section of tongue below the dorsum (coloured) p. 137	
CARCINOMA OF TONGUE - - - - -	138
A tongue drawn from the dorsal aspect and in section (coloured) p. 139	
CARCINOMA OF TONGUE - - - - -	140
A tongue removed by operation at the level of the hyoid (coloured) p. 140	
CARCINOMA OF TONGUE - - - - -	142
One half of a tongue divided by longitudinal section with the submaxillary lymphatic and salivary glands of the same side (coloured) p. 143	
CARCINOMA OF GUM - - - - -	144
The right half of an edentulous mandible with a small portion of the cheek and the sub-maxillary salivary gland (coloured) p. 144	
PAPILLOMA AND CARCINOMA OF TONGUE - - - - -	145
The left half of a tongue (coloured) p. 145	
CARCINOMA OF FLOOR OF MOUTH - - - - -	146
Anterior one-third of a tongue with the mucous membrane of the floor of the mouth and the alveolar margin of the mandible (coloured) p. 146	
CARCINOMA OF FLOOR OF MOUTH - - - - -	147
The left alveolar margin and part of the right side of a mandible with the left half of the floor of the mouth and the left half of the anterior two-thirds of the tongue (coloured) p. 147	
CARCINOMA OF LIP - - - - -	148
The lower lip and the left extremity of the upper lip with its surrounding skin (coloured) p. 149	
DIVERTICULUM OF THE PHARYNX - - - - -	150
DIVERTICULUM OF PHARYNX - - - - -	151
The viscera of the neck in sagittal section from above (coloured) p. 151	
The same from outside (coloured) p. 152	
The same in section (coloured) p. 153	
DIVERTICULUM OF PHARYNX - - - - -	154
Parts of the pharynx and œsophagus with the larynx and the upper portion of the trachea p. 155	

MALIGNANT TUMOURS OF THE PHARYNX	-	-	-	-	-	-	-	-	PAGE
CARCINOMA OF HYPOPHARYNX	-	-	-	-	-	-	-	-	156
The viscera of a neck in longitudinal section, from outside (coloured) p. 158									
The same in section (coloured) p. 159									
CARCINOMA OF SINUS PYRIFORMIS	-	-	-	-	-	-	-	-	160
A pharynx, larynx, and base of tongue (coloured) p. 161									
CARCINOMA OF ARY-EPIGLOTTIC FOLD	-	-	-	-	-	-	-	-	162
A pharynx with the tongue and larynx (coloured) p. 163									
SARCOMA OF PHARYNX	-	-	-	-	-	-	-	-	164
A pharynx with the larynx and base of the tongue, opened from behind (coloured) p. 165									
DIVERTICULUM OF THE ŒSOPHAGUS (TRACTION DIVERTICULUM)	-	-	-	-	-	-	-	-	166
DIVERTICULUM OF ŒSOPHAGUS	-	-	-	-	-	-	-	-	166
An œsophagus with the larynx, trachea, and part of the right lung p. 167									
DIVERTICULUM OF ŒSOPHAGUS	-	-	-	-	-	-	-	-	168
Part of an œsophagus with the bifurcation of the trachea p. 168									
CONGENITAL MALFORMATIONS OF THE ŒSOPHAGUS	-	-	-	-	-	-	-	-	169
CONGENITAL ATRESIA OF ŒSOPHAGUS (TRACHEO-ŒSOPHAGEAL FISTULA)	-	-	-	-	-	-	-	-	170
The tongue, larynx, œsophagus, trachea, and stomach of an infant (coloured) p. 170									
CONGENITAL STRICTURE OF ŒSOPHAGUS	-	-	-	-	-	-	-	-	171
A larynx and trachea with the lower part of the pharynx and the corresponding portion of the œsophagus p. 171									
CARDIO-SPASM	-	-	-	-	-	-	-	-	172
CARDIO-SPASM	-	-	-	-	-	-	-	-	173
The lower part of an œsophagus opened out with a portion of the cardiac end of the stomach, parts of the right lung, trachea, and aorta (coloured) p. 174									
SIMPLE TUMOURS OF THE ŒSOPHAGUS	-	-	-	-	-	-	-	-	175
BRANCHIAL CYST	-	-	-	-	-	-	-	-	176
The viscera of a neck with the pharynx and œsophagus opened from behind p. 176									
FIBROMYOMA OF ŒSOPHAGUS	-	-	-	-	-	-	-	-	177
The viscera of a neck. The pharynx and œsophagus have been opened from behind p. 177									
CARCINOMA OF THE ŒSOPHAGUS	-	-	-	-	-	-	-	-	178
CARCINOMA OF ŒSOPHAGUS	-	-	-	-	-	-	-	-	179
A pharynx and œsophagus with the tongue, larynx, and trachea (coloured) p. 179									
CARCINOMA OF ŒSOPHAGUS	-	-	-	-	-	-	-	-	180
Four sections of an œsophagus, trachea, thyroid, and surrounding tissues (coloured) p. 181									
CARCINOMA OF ŒSOPHAGUS (PERFORATING TRACHEA)	-	-	-	-	-	-	-	-	182
The upper third of an œsophagus with the larynx and trachea (coloured) p. 183									
CARCINOMA OF ŒSOPHAGUS (PERFORATING TRACHEA)	-	-	-	-	-	-	-	-	184
An œsophagus with the larynx and trachea (coloured) p. 185									
CARCINOMA OF ŒSOPHAGUS	-	-	-	-	-	-	-	-	186
Part of an œsophagus with the lower end of the trachea and the arch of the aorta (coloured) p. 187									
CARCINOMA OF ŒSOPHAGUS (PERFORATING AORTA)	-	-	-	-	-	-	-	-	188
Part of an œsophagus with the bifurcation of the trachea, the arch and part of the descending aorta (coloured) p. 189									
CARCINOMA OF ŒSOPHAGUS (PERFORATING BRONCHUS)	-	-	-	-	-	-	-	-	190
An œsophagus with the tongue, larynx, and trachea (coloured) p. 191									
Back view (coloured) p. 192									

## EXOPHTHALMIC GOITRE.

The thyroid and thymus glands together with the larynx and a part of the trachea.

The thyroid gland is uniformly enlarged and its consistence is firm. The cut surface of the right lobe shows a solid, fleshy structure. The vessels are enlarged. The enlarged thymus gland is 10 cm. long.

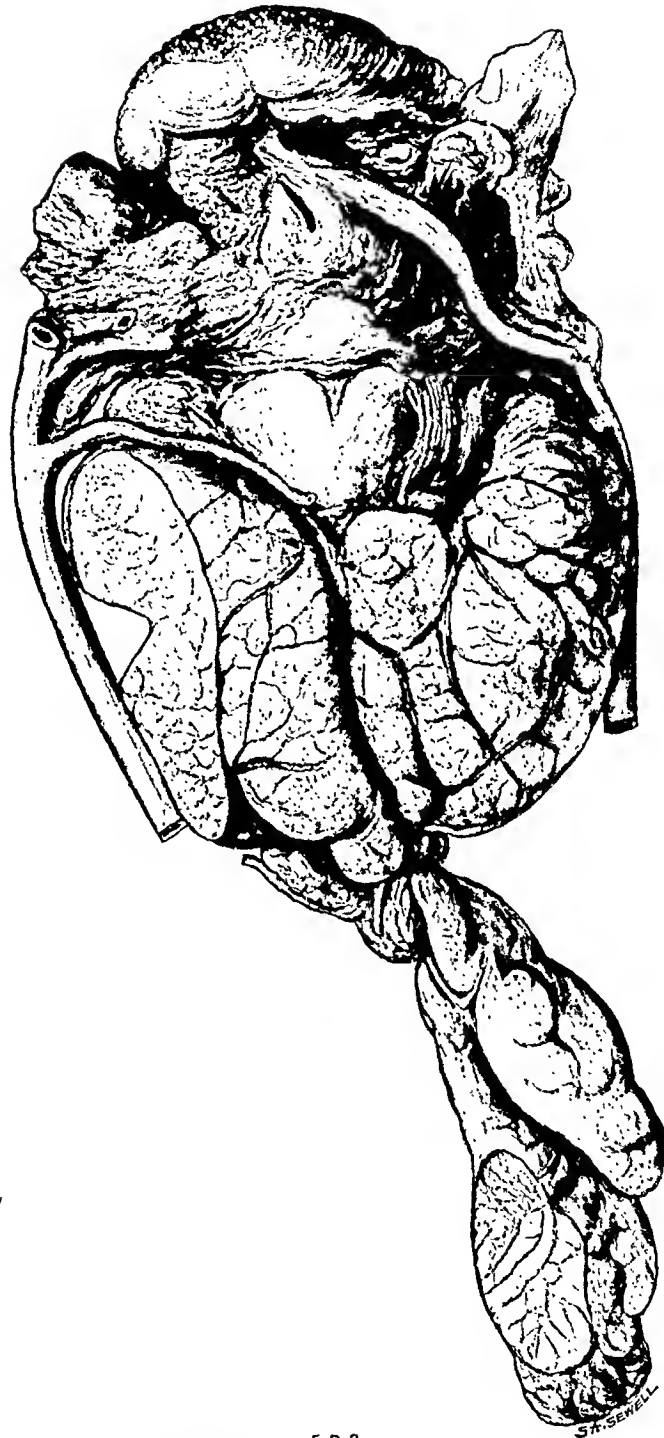
*Museum of University College Hospital, 5.D.3*

CLINICAL HISTORY.—The patient was a single nursemaid, aged 22, in whose family history there is no mention of goitre. Her illness began with palpitation two months before admission to hospital. For six weeks there had been swelling of the neck and prominence of the eyes.

On examination there was bilateral exophthalmos, the left eye being much more prominent than the right. The thyroid was enlarged and pulsated visibly. There was no enlargement of the lymph glands of the neck, but the tonsils were swollen. Her hands were tremulous. The pulse-rate was 130 on admission and rose to 210 before death. The heart was not enlarged. There was an irregular fever rising to 103° and severe headache. Vomiting began soon after admission and remained a constant feature until her death four months after the onset of the illness. Shortly before death there was slight œdema of the legs. The urine was normal.

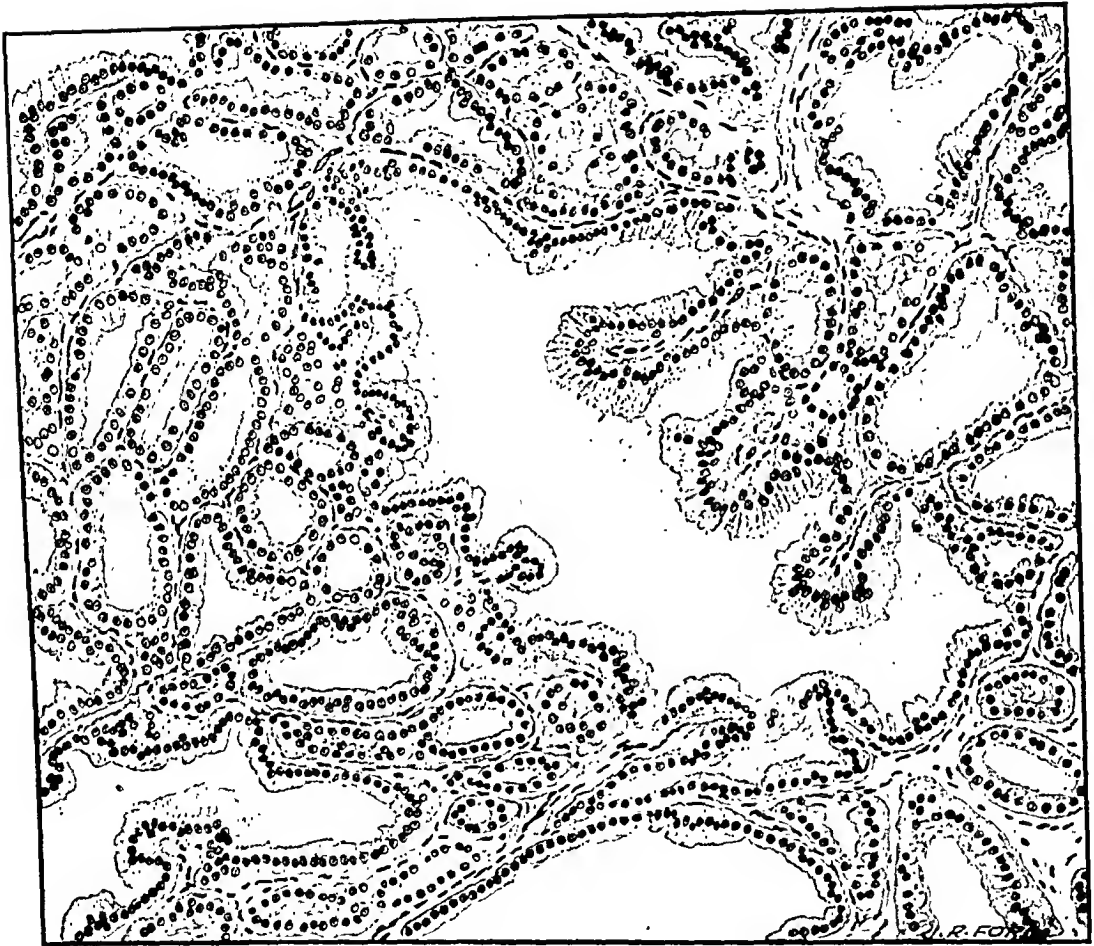
AUTOPSY.—Apart from the enlargement of the thyroid and thymus no special changes are noted in the organs.

# EXOPHTHALMIC GOITRE.



MUSEUM OF UNIVERSITY COLLEGE HOSPITAL, 5.D.3

## EXOPHTHALMIC GOITRE.



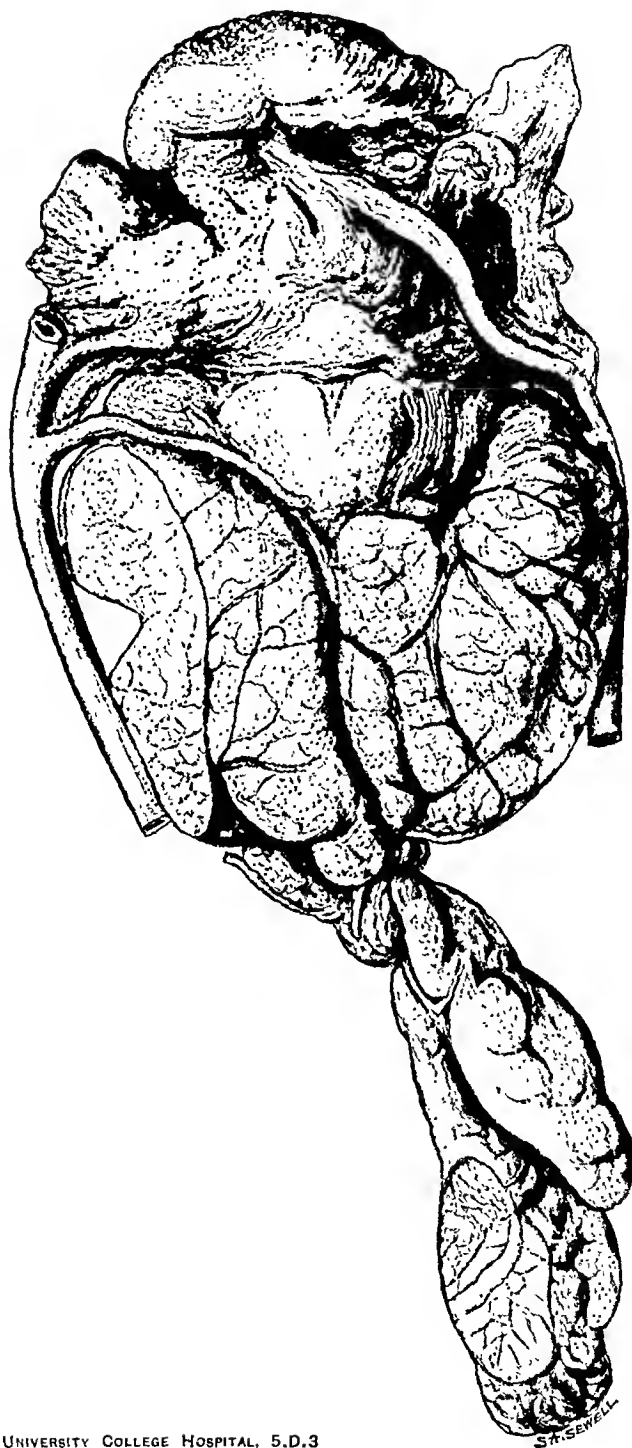
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MUSEUM OF UNIVERSITY COLLEGE HOSPITAL, 5.D.3

**MICROSCOPIC STRUCTURE.**—The alveoli of the thyroid are irregular in size and shape. They are lined by a columnar epithelium which, at many points, has proliferated so as to fill the whole or part of the alveoli. There is little colloid. The thymus retains its normal structure.

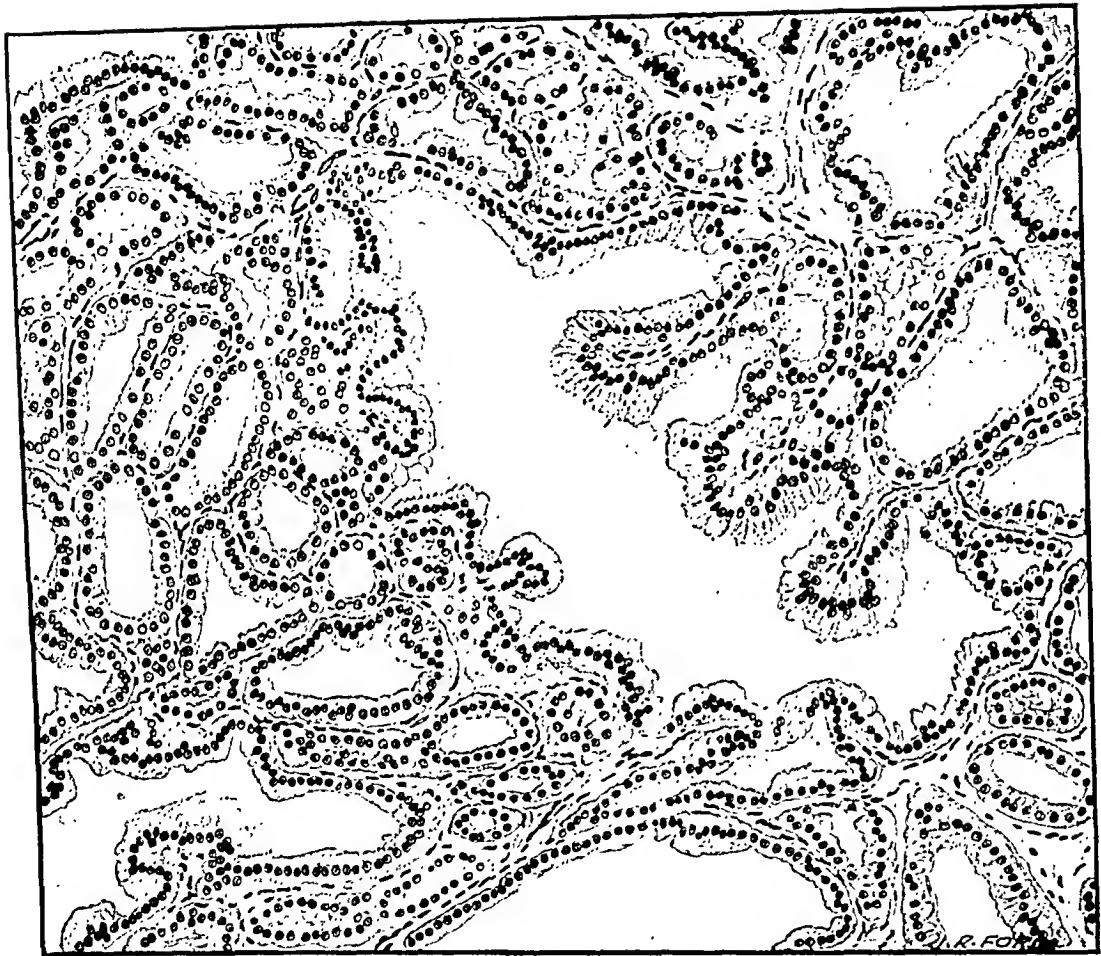


# EXOPHTHALMIC GOITRE.



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## EXOPHTHALMIC GOITRE.



x 200

MUSEUM OF UNIVERSITY COLLEGE HOSPITAL, 5.D.3

**MICROSCOPIC STRUCTURE.**—The alveoli of the thyroid are irregular in size and shape. They are lined by a columnar epithelium which, at many points, has proliferated so as to fill the whole or part of the alveoli. There is little colloid. The thymus retains its normal structure.

## XXIX. CHRONIC THYROIDITIS.

**Riedel's Disease** (*Lymphadenoid Goitre ; Woody Thyroid*).—In this rare disease, which affects both sexes, the whole or part of the thyroid becomes converted into a smooth hard mass which retains the shape of the gland with a varying increase in bulk. A preliminary enlargement may be followed by shrinking later. The shrinking is due to fibrosis within the gland, and by extension of the fibrotic process the thyroid may become fixed to other structures of the neck, trachea, carotid sheath, etc., but usually remains free from the skin. The disease may end in death by suffocation. To the naked eye the cut surface of the gland is smooth, white or buff-coloured, and opaque. Microscopically, the normal colloid vesicles are obscured by a diffuse round-celled infiltration in which many lymph follicles are formed. In the later stages there is fibrosis with compensatory overgrowth of undamaged areas of thyroid tissue. The extra-capsular extension of the disease takes the form of invasion of the cellular tissue of the neck by a dense fibrous tissue infiltrated with round cells.

**Tuberculosis.**—Miliary tubercles may appear in the thyroid during the terminal phase of generalized tuberculosis. Nodular tuberculosis is a rare disease which owes its importance chiefly to the clinical resemblance which it may bear to malignant disease in the early stage, when the nodules are hard and fixed by inflammatory infiltration. In the stage of caseation or in the presence of sinuses the diagnosis is no longer liable to confusion.

**Syphilis.**—Gumma of the thyroid gland is very rare. It affects goitrous rather than normal thyroids, and in some cases appears to have arisen as an extension of perichondritis of the thyroid cartilage.

## CHRONIC THYROIDITIS.

(RIEDEL'S DISEASE.)

The cut surface of part of a thyroid gland drawn from behind.

The whole gland is enlarged, the left lobe more than the right, and has a yellowish white, fleshy appearance. The external surface is slightly nodular, and on the cut surface there is a faint network of fibrous tissue corresponding to the divisions between the nodules. The capsule has a few filmy adhesions on its surface.

*Museum of University College Hospital, 676/29*

CLINICAL HISTORY.—The patient was a calm housewife, aged 45, who complained of swelling of the neck, shortness of breath, and cough. The swelling had been present for three years and was growing rapidly.

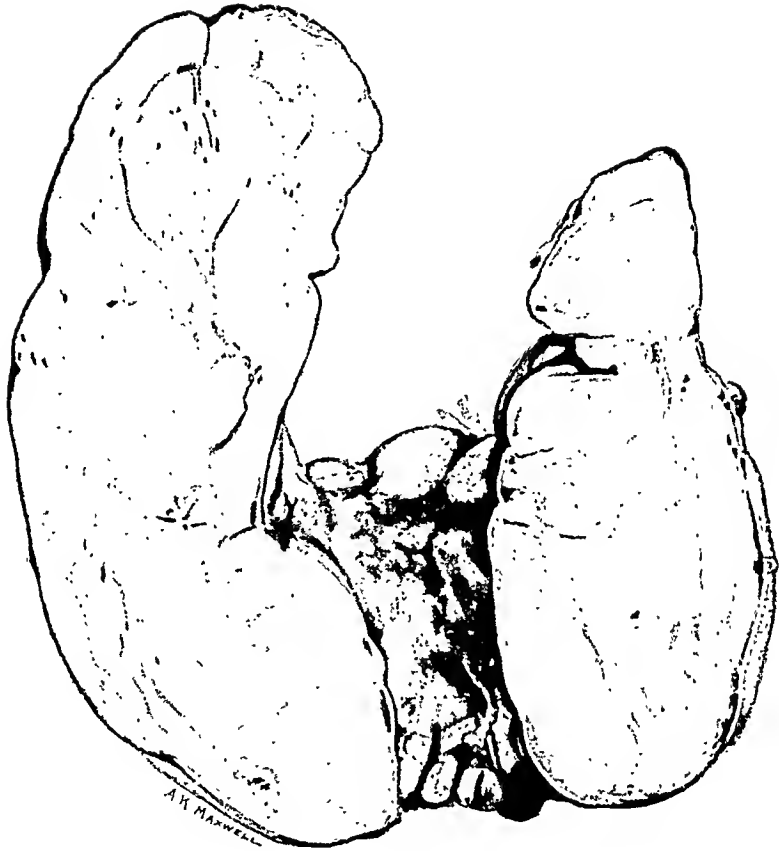
On examination there was a nodular enlargement of the whole thyroid which extended down behind the sternum and had pushed both carotids back as far as the posterior borders of the sternomastoid muscles. The thyroid was not fixed to surrounding structures. The lymph glands of the neck were not enlarged. There was stridor but no toxic symptoms.

X-ray showed bilateral compression of the trachea.

The greater part of the thyroid was removed by operation, and convalescence was uneventful.

# CHRONIC THYROIDITIS.

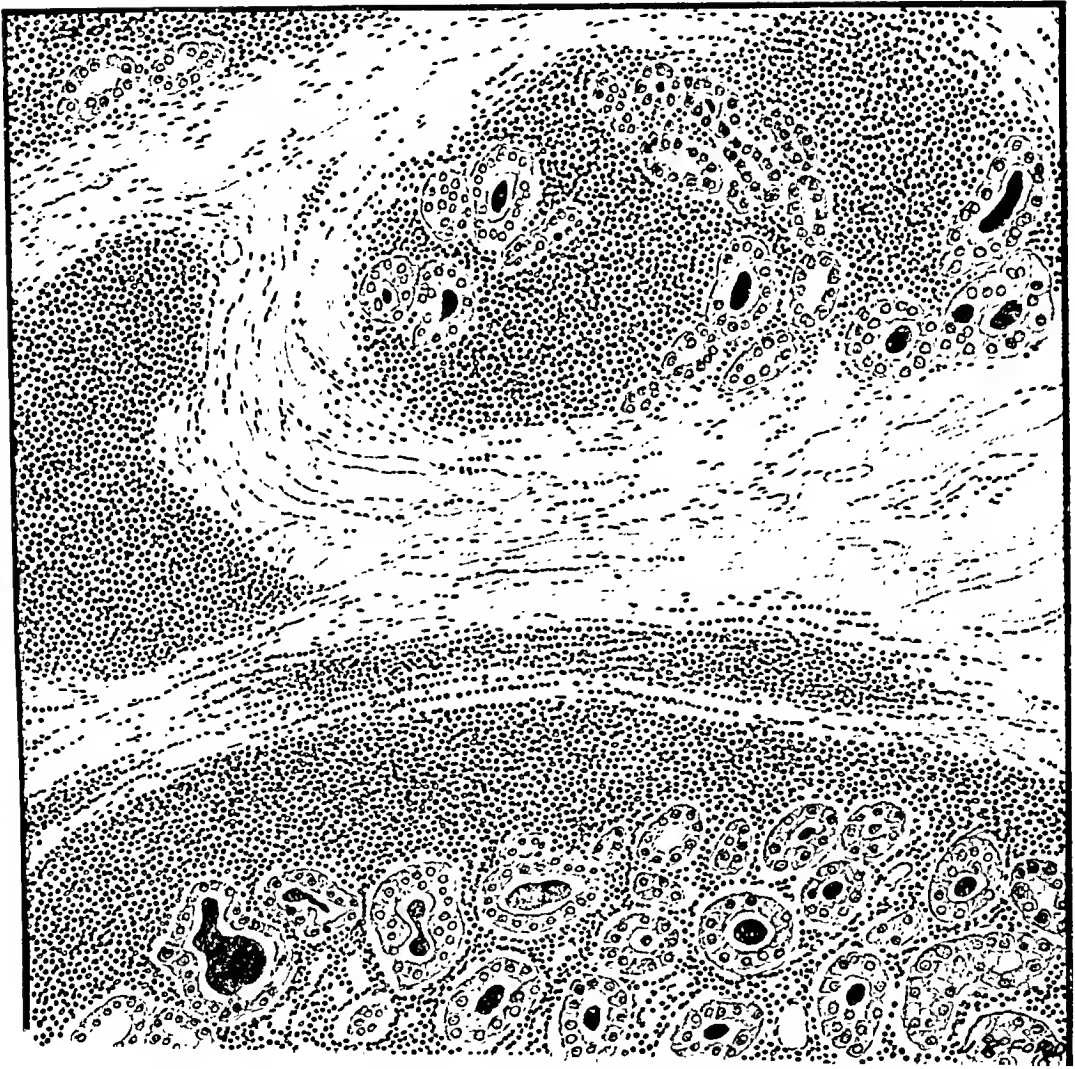
(RIEDEL'S DISEASE.)



MUSEUM OF UNIVERSITY COLLEGE HOSPITAL, 676/29

## CHRONIC THYROIDITIS.

(RIEDEL'S DISEASE.)



MUSEUM OF UNIVERSITY COLLEGE HOSPITAL, 676/29

× 130

**MICROSCOPIC STRUCTURE.**—The thyroid vesicles are greatly reduced in numbers, small, and contain little colloid. The greater part of the section is made up of masses of lymphoid tissue between which are thick strands of fibrous tissue.

## CHRONIC THYROIDITIS.

(RIEDEL'S DISEASE.)

A thyroid gland, part of which has been removed.

The thyroid is uniformly enlarged to a moderate degree and the vessels beneath its capsule are small and few. The cut surface is pale and fibrous.

*Museum of the College of Medicine, University of Durham, Newcastle-upon-Tyne, 540/1*

MICROSCOPIC STRUCTURE.—There is considerable atrophy of the thyroid vesicles, accompanied by fibrosis and extensive infiltration by mononuclear and lymphoid cells.

CLINICAL HISTORY.—The patient was a woman, aged 58, who was admitted to hospital suffering from an enlarged thyroid gland and dyspnoea. Four years before she had noticed a swelling which was at times painful. Eighteen months before she began to have difficulty in breathing, and this had increased with the enlargement of the thyroid. At times her voice became husky, and occasionally it failed. There was no difficulty in swallowing. Her memory had become very bad.

On examination there was slight myxœdema. The thyroid was uniformly enlarged, but moved freely on swallowing. It was hard, and in places stony hard. There was slight dyspnoea and huskiness of the voice, but no evidence of pressure on vessels. No glandular enlargement or spread into the mediastinum.

At operation the whole gland was removed. Thyroid was administered for a time, but the dose was gradually diminished, and it was discontinued about six months after operation without detriment. She was then in good health apart from her slight myxœdema.

(*A. F. Bernard Shaw and R. P. Smith, British Journal of Surgery, 1925, xiii, 99.*)

CHRONIC THYROIDITIS.  
(RIEDEL'S DISEASE.)



MUSEUM OF THE COLLEGE OF MEDICINE, UNIVERSITY OF DURHAM, NEWCASTLE-UPON-TYNE, 540/1



### XXX. MALIGNANT DISEASE OF THE THYROID GLAND.

MALIGNANT tumours of the thyroid may, but do not often, grow in a previously healthy gland. They commonly arise in a nodular goitre of some standing. The disease is about twice as common in women as in men. While a malignant tumour is still confined within the capsule of the thyroid the only special signs which can be attributed to its malignant nature are rapid growth and increasing hardness. As soon as it penetrates the capsule, a third sign is added—loss of mobility. This is usually produced by fixation to the trachea. If the growth lies at the back of the gland, recurrent laryngeal paralysis is added to the early signs. As soon as the tumour has grown definitely beyond the capsule of the thyroid, its rapid increase in size and progressive attachment to the other structures of the neck make the diagnosis obvious. Secondary deposits are carried by the lymphatics to the glands of the jugular chain and posterior triangle and to the thymus. More distant metastases show a definite preference for bones.

While the different histological types of malignant goitre cannot accurately be correlated with their clinical signs, a broad generalization may be made that the more closely a tumour resembles a simple goitre in its microscopic appearance, the less malignant will it be. Among metastasizing neoplasms of slow growth are the malignant colloid goitre, in which both primary and secondary tumours resemble a simple colloid goitre, the proliferating adenoma (Langhans), and the papilliferous cystic adenoma. All these are rare. Ordinary spheroidal or cubical-celled carcinoma is the usual form of malignant disease of the thyroid, and presents no special differences from similar growths in other glands. Sarcoma cannot, with certainty, be differentiated from carcinoma in the thyroid, on account of the constancy with which masses of proliferating epithelium are mixed with the proliferating connective-tissue elements.

Secondary carcinoma is rarely found in the thyroid except in the case of direct extension from a primary growth of the cervical oesophagus. This appears first as an indefinite induration of the back of one lobe and spreads forwards until it has involved a large part of the gland. Its appearance in the thyroid often precedes that of a clinically recognizable stricture of the oesophagus.

## CARCINOMA OF THYROID.



The right lobe of a thyroid gland in section.

In its lower part is an encapsuled adenoma containing a slit-like cyst. Above and to the right of this is a crescentic, yellowish-white carcinoma, the surface of which is depressed below the general level of the section by contraction of its fibrous stroma. A layer of compressed thyroid tissue surrounds the whole.

*Museum of University College Hospital, 13.D.5*

**CLINICAL HISTORY.**—The patient was a man, aged 59, who had noticed a painful lump on the right side of his neck for eleven days. He had slight difficulty in swallowing. In the right lobe of the thyroid was a hard, tender mass fixed to the trachea, which was pushed slightly to the left. The vocal cords were normal. Soft enlarged glands were present on both sides of the neck.

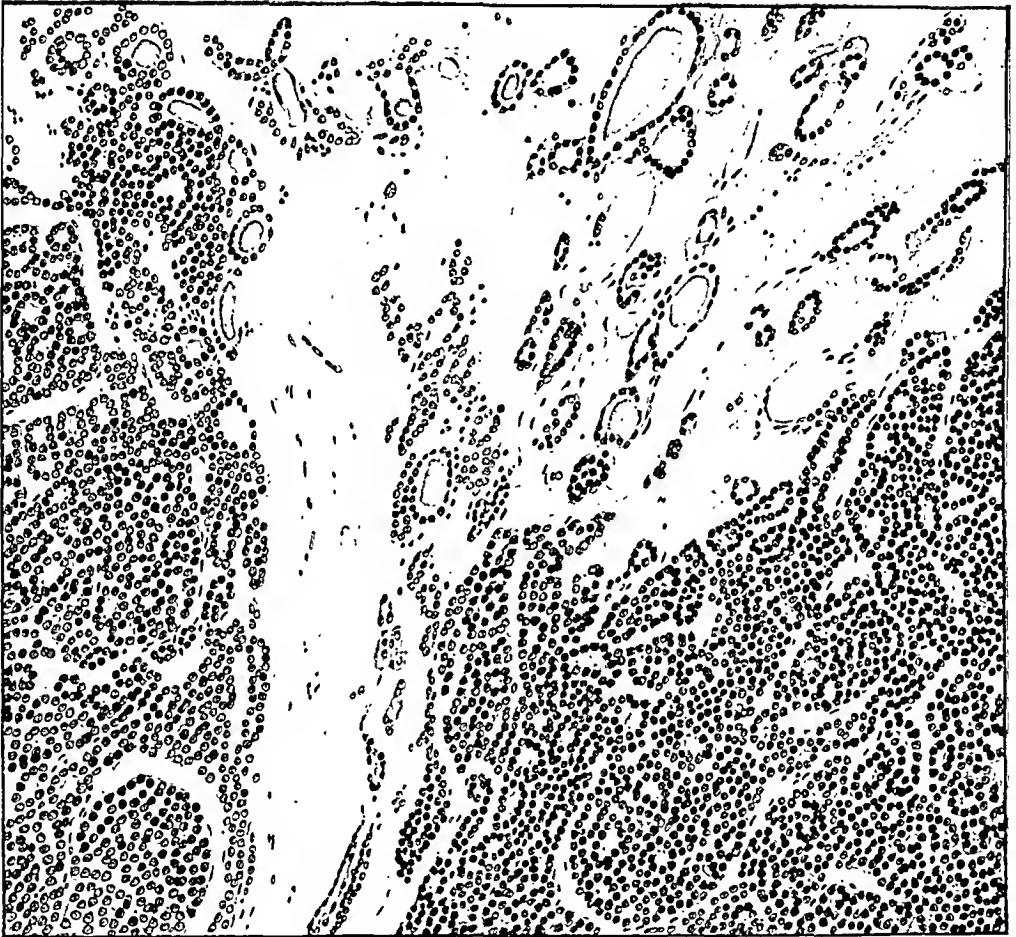
**Operation:** right hemithyroidectomy. The wall of the pharynx was infiltrated by growth, and lymphatic vessels distended with growth were seen running to the glands on the internal jugular vein.

One month after operation there was a large mass in the neck producing symptoms of pressure on trachea, recurrent laryngeal nerve, and oesophagus. Death occurred from asphyxia two months after the onset of the disease.

**AUTOPSY.**—Secondary deposits in the lungs, pleura, and vertebral column.

# CARCINOMA OF THYROID.

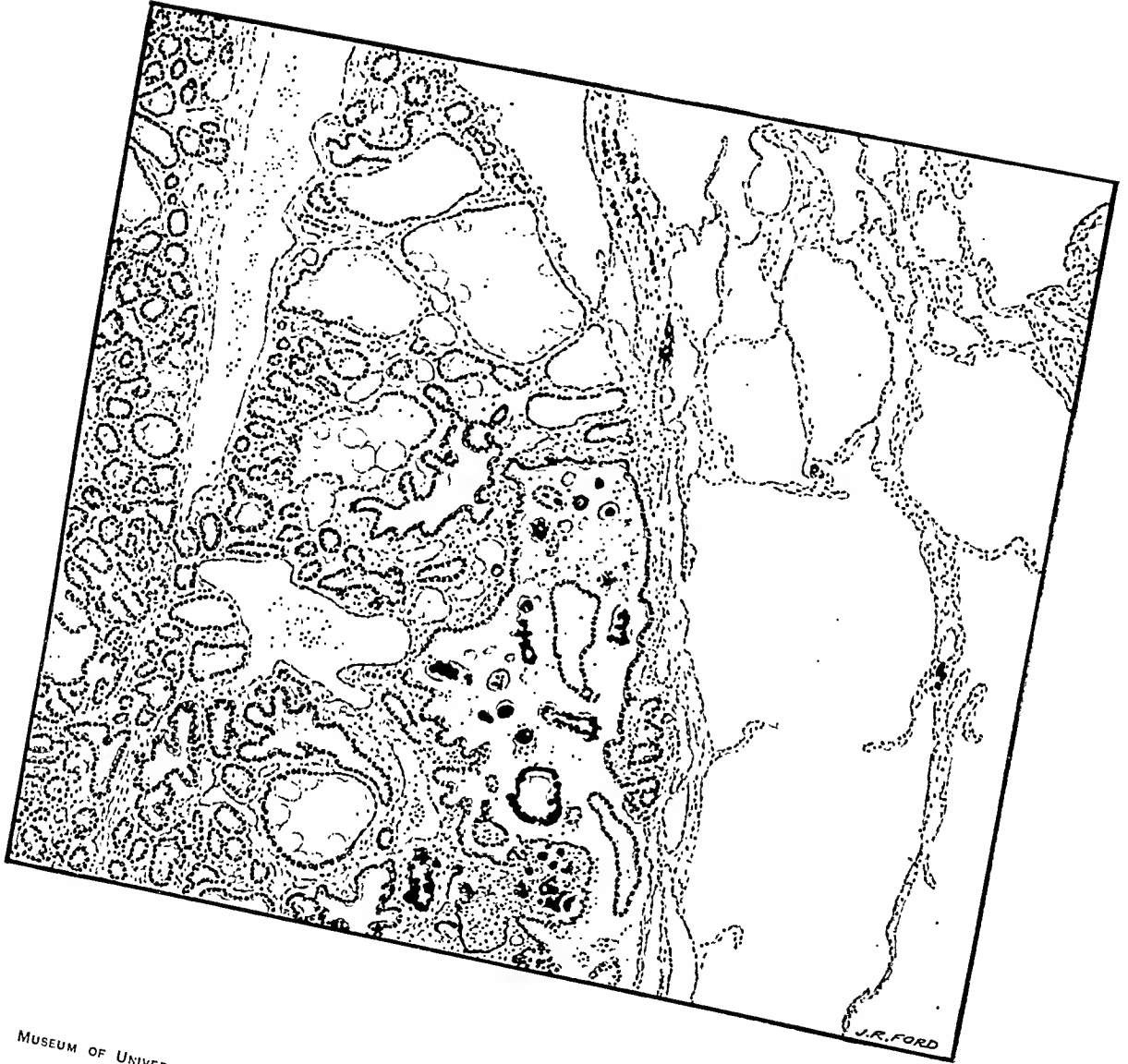
(PRIMARY GROWTH.)



× 180

MUSEUM OF UNIVERSITY COLLEGE HOSPITAL, 13.D.5

CARCINOMA OF THYROID.  
(SECONDARY GROWTH IN LUNG.)



× 76

MUSEUM OF UNIVERSITY COLLEGE HOSPITAL, 13.D.5

## CARCINOMA OF THYROID.

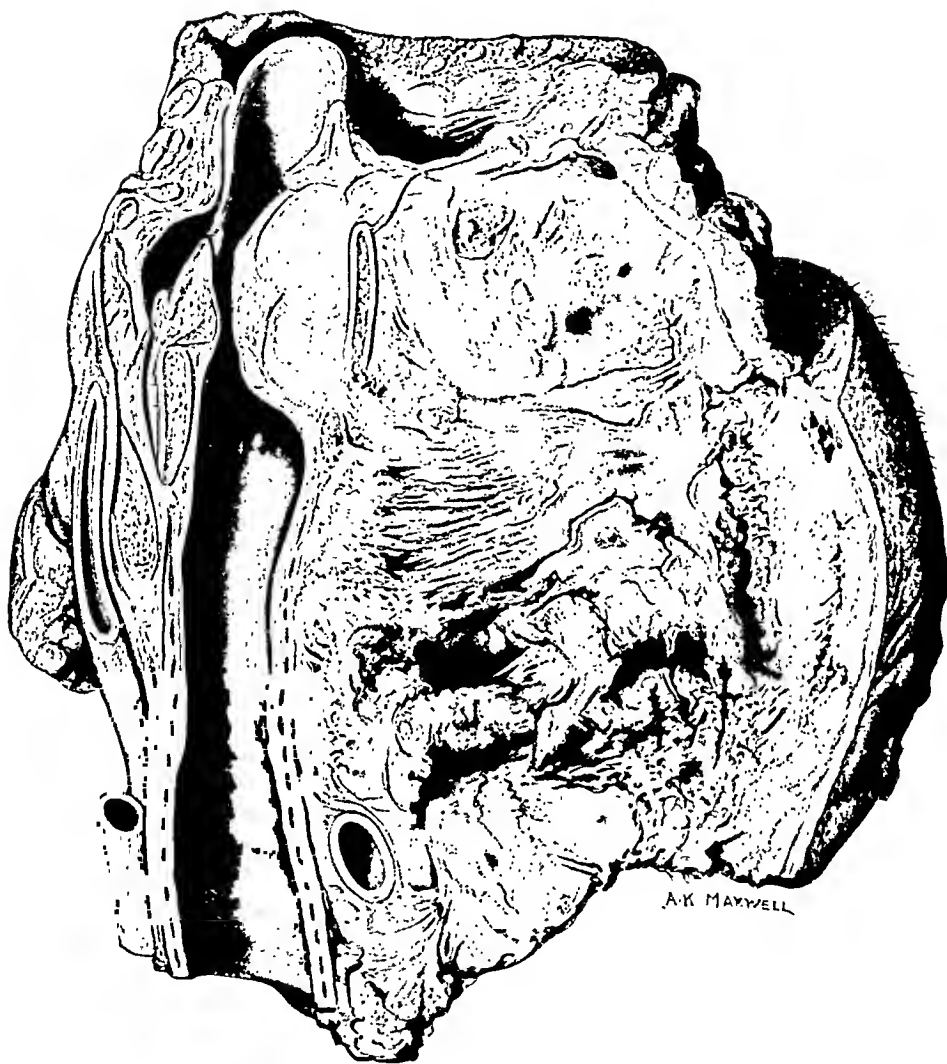
(LOCAL RECURRENCE.)

The anterior half of a coronal section through the neck.

To the right of the trachea the neck is distended by a mass of growth with hæmorrhage in its centre. The growth has invaded the larynx above and below the thyroid cartilage and is fungating through the scar of operation.

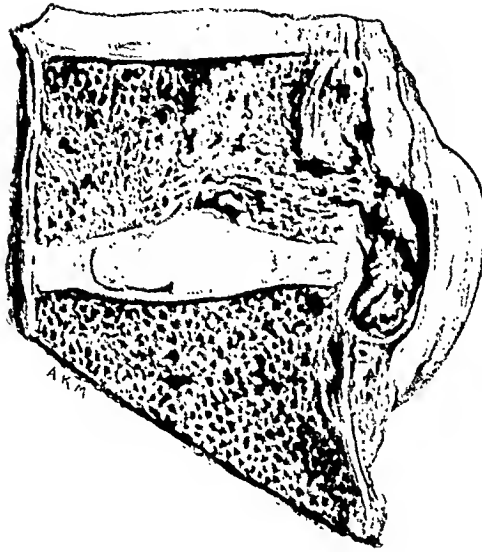
*Museum of University College Hospital, 13.D.6*

CARCINOMA OF THYROID.  
(LOCAL RECURRENCE.)



MUSEUM OF UNIVERSITY COLLEGE HOSPITAL, 13.D.6

CARCINOMA OF THYROID.  
(SECONDARY GROWTH IN BONE.)

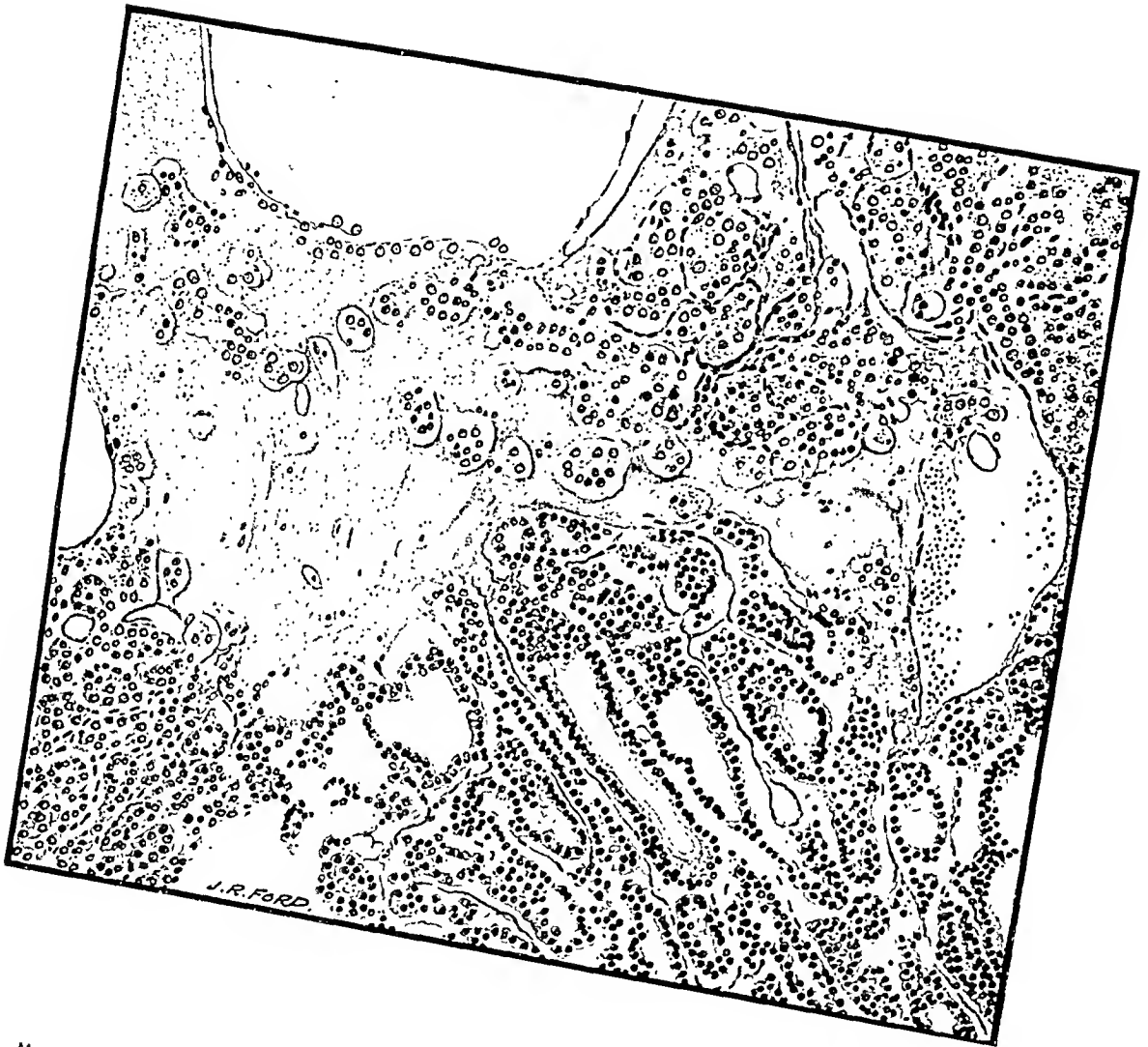


Portions of two lumbar vertebræ in sagittal section.

On the antero-lateral surface of the body of the upper vertebra is a nodular mass of growth, which had infiltrated the bone as far back as the spinal canal.

*Museum of University College Hospital, 13.D.7*

CARCINOMA OF THYROID.  
(SECONDARY GROWTH IN BONE.)

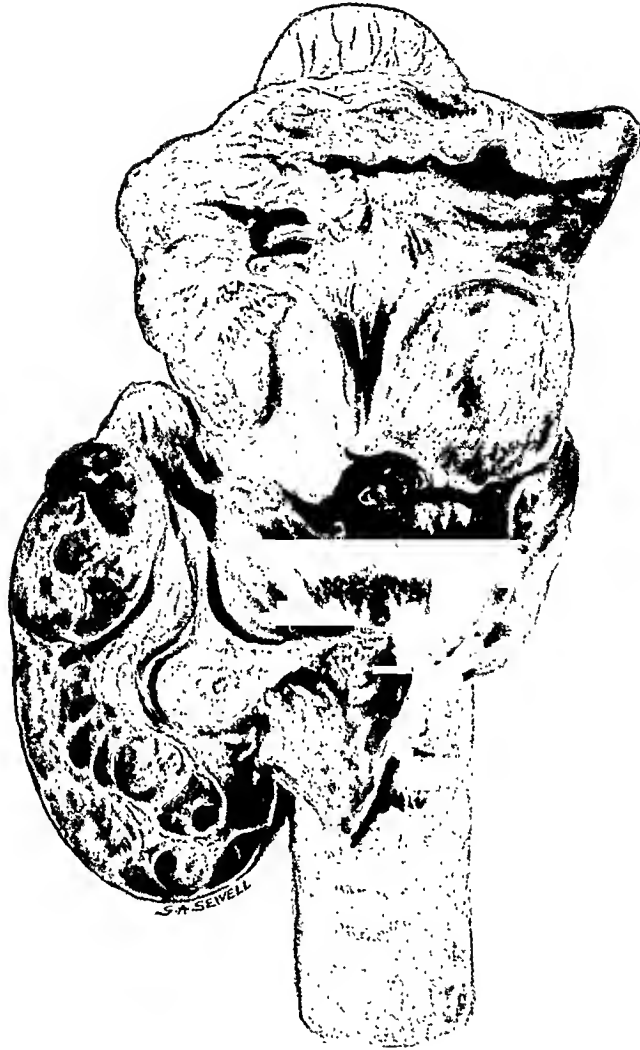


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MUSEUM OF UNIVERSITY COLLEGE HOSPITAL, 13.D.7



## CARCINOMA OF THYROID.



A larynx and trachea with a portion of the thyroid gland drawn from the front.

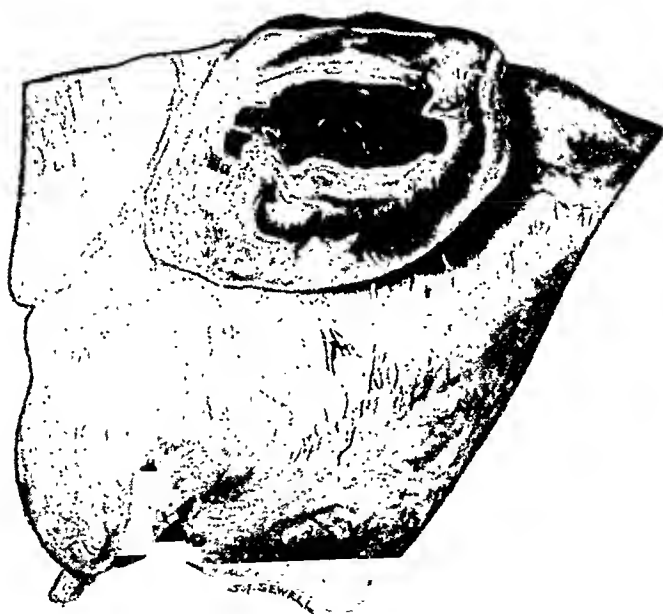
The right lobe of the thyroid has been divided by coronal section. It contains several colloid nodules. On its tracheal aspect is a white growth which is hard and depressed below the surface of the section.

*Museum of the College of Medicine, University of Durham, Newcastle-upon-Tyne, C.4719*

MICROSCOPIC STRUCTURE.—Carcinoma.

No clinical history.

CARCINOMA OF THYROID.  
(SECONDARY GROWTH IN SKULL.)



Part of the left side of a skull.

Above and behind the mastoid process is a tumour which projects above the surface and has broken through the expanded bone in the centre.

The section shows that the whole thickness of the skull has been destroyed, and that the growth has displaced but has not penetrated the dura.

*Museum of the College of Medicine, University of Durham, Newcastle-upon-Tyne, C.4719*

## PROLIFERATING ADENOMA OF THYROID.

(TYPE LANGHANS.)

The right lobe of a thyroid gland.

The outer surface is nodular and is surrounded by a fibrous capsule. The section shows a solid, yellowish-white, and vascular growth.

*Museum of University College Hospital, 9.D.3*

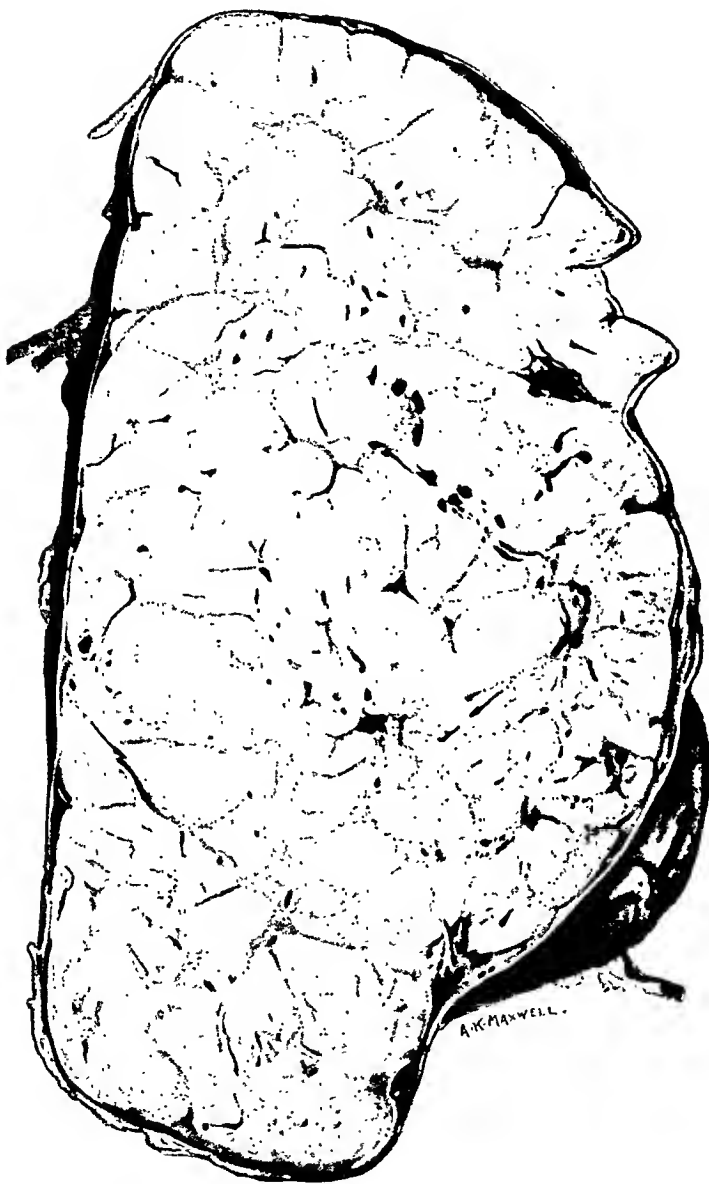
MICROSCOPIC STRUCTURE.—The alveoli are densely packed with epithelial cells. Here and there is a colloid vesicle. There is a well-defined capsule.

CLINICAL HISTORY.—The patient was a man, aged 52, who had had a swelling of the neck for 15 years without any symptoms.

On examination there was a large, collar-like mass in the position of the thyroid. It was hard, solid, and well defined. The lymph glands were not enlarged.

The mass was removed by operation, at which the left lobe of the thyroid was found to be normal. The patient remained free from recurrence for eleven months, but died of a 'throat complaint' seven years after operation.

PROLIFERATING ADENOMA OF THYROID.  
(TYPE LANGHANS.)



MUSEUM OF UNIVERSITY COLLEGE HOSPITAL, 9.D.3

## CARCINOMA OF THYROID.

One half of a vertical section through the right lobe of the thyroid gland and the trachea.

The thyroid is enlarged by a carcinoma which is encapsuled except where it is invading the trachea. The growth is of an opaque white colour and is degenerating in its central and lower parts.

The trachea is curved round the tumour and is narrowed in its antero-posterior diameter, but the mucous membrane is intact.

*Hunterian Museum, R.C.S., 7224.1*

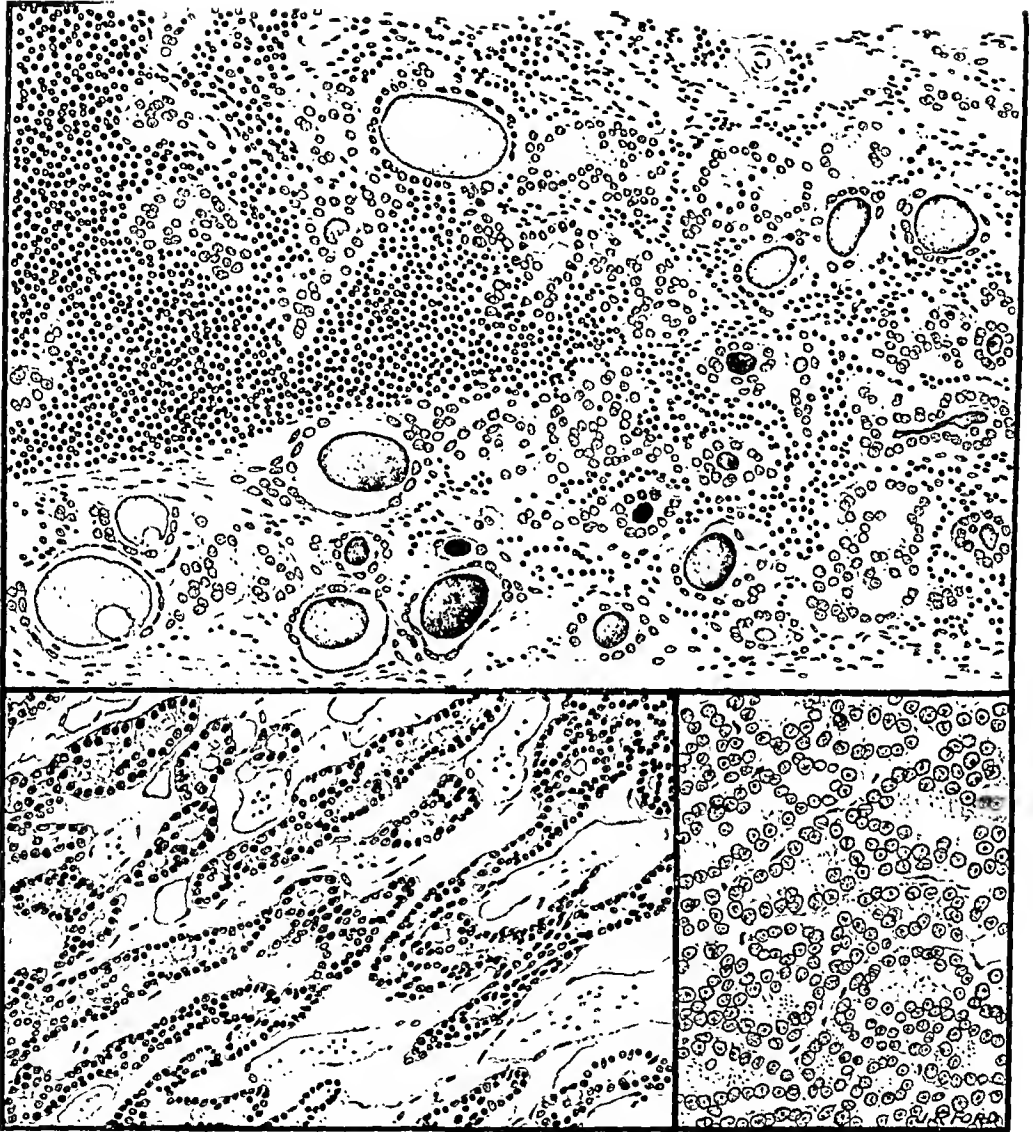
CLINICAL HISTORY.—The patient was a woman, aged 58. Two years before her death her voice became altered and a swelling appeared in the neck.

# CARCINOMA OF THYROID.



HUNTERIAN MUSEUM, R.C.S., 7224.1

## CARCINOMA OF THYROID.



× 200

HUNTERIAN MUSEUM, R.C.S.. 7224.1.

**MICROSCOPIC STRUCTURE.**—The growth is a spheroidal-celled carcinoma which is made up of narrow strands of epithelial cells lying in a fibrous stroma.

## XXXI. INFLAMMATION, LEUCOPLAKIA, AND ULCERATION OF THE TONGUE.

**Inflammation.**—The tongue readily becomes œdematous as a consequence of any inflammatory condition within the mouth, whether this be general as in mercurial stomatitis, or local as in ulceration of the gum around a tooth with infected socket.

*Acute superficial glossitis* is caused by scalding or caustic fluids, or occurs as part of a general stomatitis, e.g., mercurial.

*Acute deep (parenchymatous) glossitis* is associated with oral sepsis, and may occasionally be started by a penetrating wound. The tongue is swollen by œdema and may develop one or more abscesses.

**Chronic Superficial Glossitis: Leucoplakia.**—Chronic superficial glossitis is a common disease caused by irritants of low intensity acting over a long period of time. The chief of these are septic teeth and syphilis, with tobacco and alcohol as adjuvants. In the usual form the papillæ disappear from irregular areas of the mucous membrane and a thin, shiny, red epithelium remains. This becomes horny and sodden, and forms smooth, white patches. The condition is then known as leucoplakia. Beneath the epithelium there is a round-celled infiltration of the mucous membrane with development of scar tissue which contracts to form fissures on the surface of the tongue. Ulceration occurs both in the patches of atrophic epithelium and at the bottom of the fissures.

Less commonly chronic superficial glossitis assumes an hypertrophic form with overgrowth of the filiform papillæ and thickening of the epithelium. The long papillæ are blackened by bacteria (*hairy tongue, nigrities*) or form warty patches on the dorsum.

Leucoplakia is seen not only on the tongue but also on the mucous membrane of the cheek and lips. On the cheek it appears as a strip running backwards from the angle of the mouth corresponding to the area in contact with the teeth.

The chief practical importance of chronic superficial glossitis lies in its tendency to be followed by carcinoma, which may arise in a smooth or warty patch or in an ulcer or fissure.

*Erythema migrans (geographical tongue)* is a form of chronic superficial glossitis which occurs in young children of poor health. The lesion consists in atrophy of the filiform papillæ and their replacement by a thin, smooth layer of epithelium. The process starts simultaneously at several points and spreads centrifugally, so that the circular, red areas first produced ultimately coalesce to form an irregular pattern on the dorsum of the tongue.

**Ulcers.**—*Traumatic ulcers* are usually due to the sharp edges of cavities in carious teeth, and hence are situated on the edge of the tongue immediately opposite their obvious cause. They may also result from the pressure of



ill-fitting or rough dentures. In children with whooping-cough the frænum may become ulcerated by constant jerking against the lower incisors.

*Ulcers in chronic superficial glossitis* have already been mentioned. Induration of the edge of such an ulcer should always arouse the suspicion of carcinoma.

*Dyspeptic ulcers* are small, circular, punched-out ulcers occurring in groups near the tip of the tongue.

*Herpetic ulcers* result from infection of the vesicles in herpes of that part of the fifth nerve which supplies the tongue.

Ulceration of the tongue also occurs in mercurial stomatitis.

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## ACUTE PARENCHYMATOUS GLOSSITIS.

### (ABSCESS OF TONGUE.)

A tongue with the larynx and pharynx. The pharynx has been opened from the back.

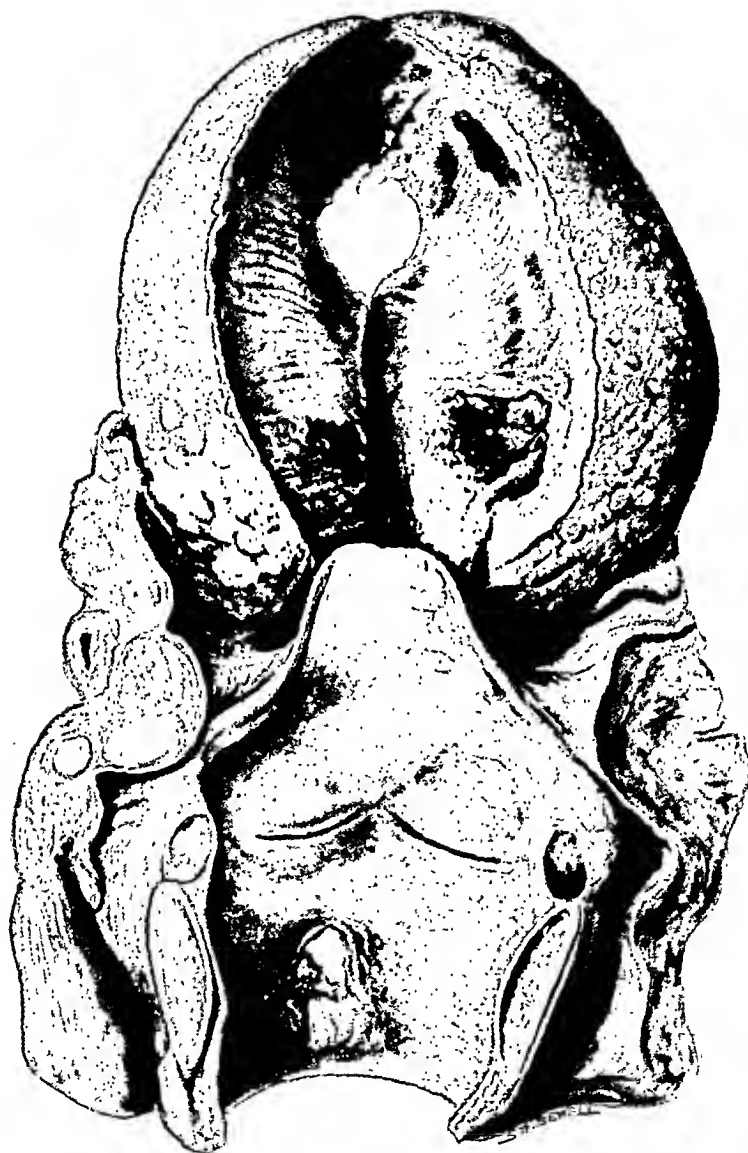
The tongue is enlarged by inflammatory exudate. There are abscess cavities at the tip and at the base. Laryngotomy has been performed.

*Museum of University College Hospital, 2.A.G.1*

CLINICAL HISTORY.—The patient was a man, aged 28. He was admitted to hospital in a state of respiratory distress with a greatly swollen tongue. There was a triangular laceration on the under surface of the tip of the tongue. This laceration was grey and septic in appearance and extended in the substance of the tongue to within a short distance of the dorsum. At the root of the tongue was an abscess cavity filled with necrotic fluid. There was no recent inflammation of the glands of the neck. He died shortly after laryngotomy had been performed.

AUTOPSY.—Septic broncho-pneumonia,

ACUTE PARENCHYMATOUS GLOSSITIS.  
(ABSCESS OF TONGUE.)



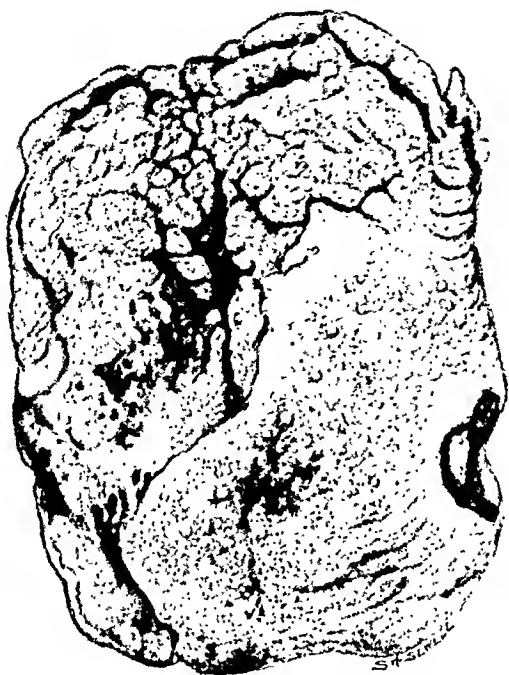
MUSEUM OF UNIVERSITY COLLEGE HOSPITAL, 2.A.G 1

## XXXII. TUBERCULOSIS AND SYPHILIS OF THE TONGUE.

**Tuberculosis.**—Tuberculosis of the tongue is probably always secondary to tuberculosis of the lung, and arises through inoculation of some chance abrasion of the mucous membrane by tuberculous sputum. The characteristic lesion is a shallow ulcer, near the tip, with sharply-defined edges which are neither raised nor indurated, and a floor formed of smooth, pale granulation tissue. This appearance is, however, so frequently dominated by the results of secondary septic infection introduced from the mouth that the clinical picture of tuberculosis of the tongue is one of great variety.

**Syphilis.**—The primary sore is occasionally seen on the tongue, and may appear as a hard chancre or an ulcer. The submaxillary and submental lymphatic glands are enlarged. In the secondary stage mucous tubercles are common and are liable to become ulcerated. In the tertiary stage chronic superficial glossitis occurs and the surface of the tongue becomes disfigured by multiple fissures in which ulceration is common. Gummata are also formed, both on the surface and in the substance of the tongue. Superficial gummata are usually multiple and affect chiefly the dorsum. They have not the characteristic relation to a source of irritation of the simple, traumatic ulcer. Gummata tend to soften and discharge on the surface of the mucous membrane, leaving a deeply excavated ulcer.

## TUBERCULOUS ULCER OF TONGUE.



A tongue drawn from the dorsum.

There is an extensive ulcer in the right half of the dorsum of the tongue, passing round beneath the tip to the left margin. The edges are not raised and the floor is formed by granulation tissue. There is no leukoplakia. The posterior part of the main ulcer is deeply fissured.

*Museum of University College Hospital, 5.A.G.2*

### MICROSCOPIC STRUCTURE.—Tubercle.

CLINICAL HISTORY.—The patient was a man, aged 54, who suffered from advanced pulmonary tuberculosis. He had had a painful tongue for two years. The tongue was enlarged, œdematous, infiltrated at the sides, and ulcerated. There were extensive lesions of both lungs and the sputum contained tubercle bacilli. The average temperature was 99° to 102°. He died shortly after admission to hospital.

AUTOPSY.—Extensive pleural adhesions. The upper lobe of the left lung was converted into a thin-walled, irregular cavity, with caseous material in its walls. The lower lobes of both lungs contained closely-packed groups of miliary tubercles without any chronic focus. The right ary-epiglottic fold was œdematous. The trachea was normal. There were tuberculous lesions in the small intestine, appendix, and kidneys.

### XXXIII. SIMPLE TUMOURS OF THE TONGUE.

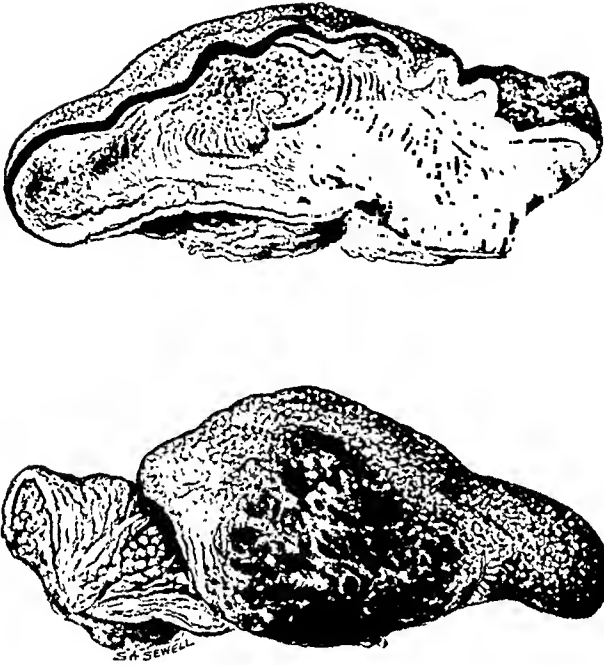
**Hæmangioma.**—Nævus of the tongue is not uncommon, and is usually of the cavernous variety. Unless large it seldom causes pain or hæmorrhage before middle age.

**Lymphangioma.**—Dilatation and overgrowth of the lymphatic tissues of the tongue is the usual cause of congenital enlargement of the tongue (*macroglossia*). The dilated lymphatics form spaces in the substance of the tongue and appear on the surface as small vesicles which discharge clear fluid.

**Papilloma** may occur as a pedunculated tumour on an otherwise normal tongue or may take the form of a warty patch in chronic superficial glossitis. It has a strong tendency to malignant degeneration.

No other simple tumour is at all common in the tongue.

## HÆMANGIOMA OF TONGUE.



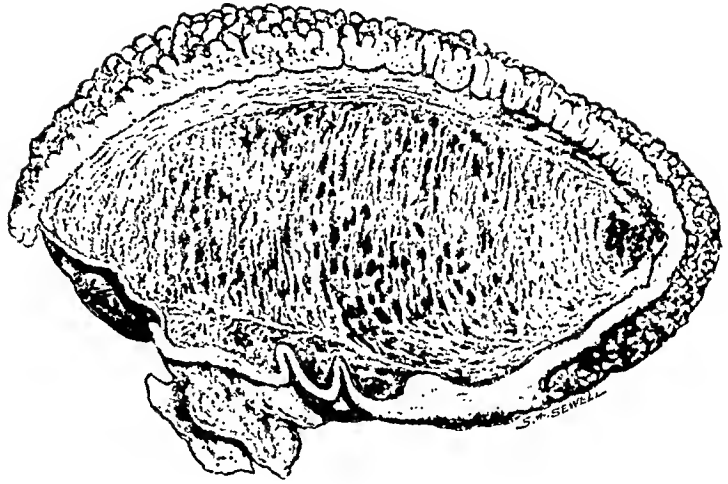
The right half of a tongue.

The anterior two-thirds of the tongue is occupied by an angioma which forms a considerable projection on the dorsum and along the right margin. The groove behind the tumour was made by a ligature.

*Hunterian Museum, R.C.S., 1434.1*

**CLINICAL HISTORY.**—The patient was a man, aged 46, who had had the condition since birth. It only began to cause inconvenience nine weeks before it was removed by operation.

## ANGIOMA OF TONGUE.



The papillæ are very prominent. The substance of the tongue is formed by a spongy mass of dilated blood-channels.

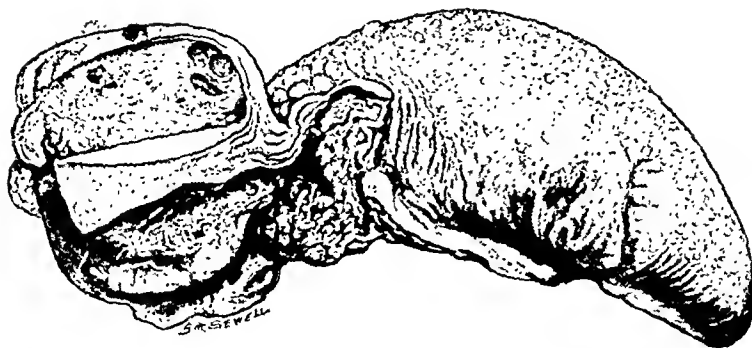
*Hunterian Museum, R.C.S., 1435.1*

**MICROSCOPIC STRUCTURE.**—The tumour consists of anastomosing channels lined by endothelium and distended with blood. They lie among the connective and muscular tissues of the tongue.

**CLINICAL HISTORY.**—The patient was a girl, aged 16, who was otherwise healthy. The swelling of the tongue was noticed at 3 years, but gave no trouble until three weeks before admission to hospital. She then became anæmic, sleepless and feverish, and the tongue increased greatly in size, so as to protrude for 4 in. from the mouth. Over the anterior part the epithelium was dry, with many points of superficial hæmorrhage. The posterior part was covered by vesicles.

The specimen illustrated was removed by operation.

## ENDOTHELIOMA OF TONGUE.



The right half of a tongue.

A smoothly lobulated tumour, 3 cm. in diameter, has grown from the posterior part of the lateral border. It is covered by mucous membrane.

*Hunterian Museum, R.C.S., 1448.1*

**MICROSCOPIC STRUCTURE.**—The tumour is an endothelioma which has apparently arisen from the walls of the lymph spaces. It is not connected with the surface epithelium.

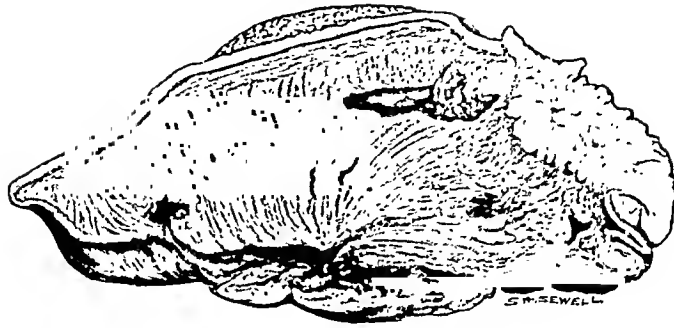
**CLINICAL HISTORY.**—The patient was a woman, aged 61, who had complained of discomfort and pain in the throat for six months. There was no difficulty in swallowing. There was enlargement of the lymph-glands beneath the angle of the jaw on the same side.

The affected half of the tongue, with the glands on the same side, was removed by operation. The enlarged glands were not examined microscopically.

The patient was free from recurrence eight years later.



## PERITHELIOMA OF TONGUE.



Part of a tongue divided by longitudinal section.

On the pharyngeal aspect of the tongue there is a slightly raised, white growth. It is superficially ulcerated, and on its deep surface has grown down into the muscle of the tongue.

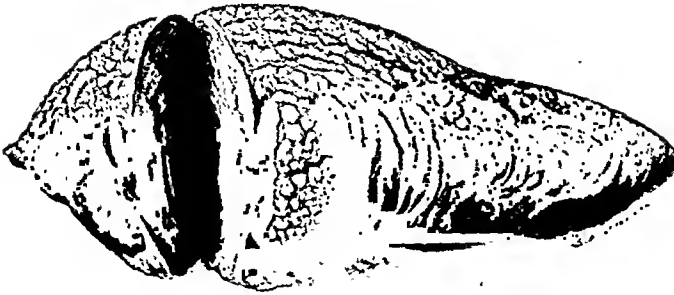
*Hunterian Museum, R.C.S., 1448.2*

**MICROSCOPIC STRUCTURE.**—The tumour is composed of endothelial cells growing around the smaller blood-vessels.

**CLINICAL HISTORY.**—The patient was a woman, aged 62, who complained of an uncomfortable feeling as if a foreign body had lodged in her throat for two months. There was no pain.

The tongue and associated lymph-glands were removed by operation. Convalescence was uneventful, and the patient was free from recurrence seven months later.

## PAPILLOMA OF TONGUE.



The right half of a tongue.

A small papillary growth projects from the lateral border about 4.5 cm. behind the tip. The growth has not invaded the muscle.

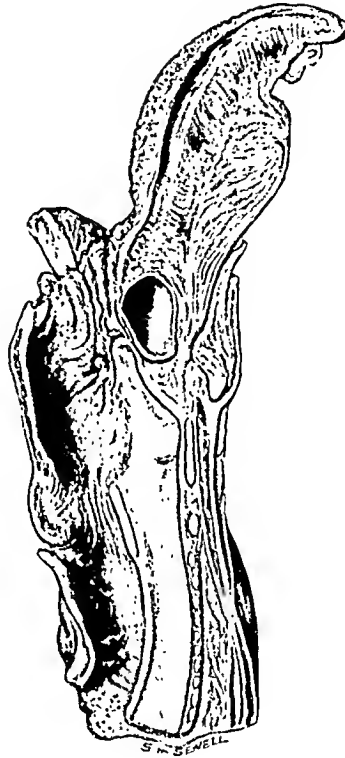
*Hunterian Museum, R.C.S., 1489.1*

MICROSCOPIC STRUCTURE.—Papilloma.

CLINICAL HISTORY.—The patient was a man, aged 49, who had had a sore tongue for six weeks. He was a light smoker. There was no enlargement of the lymph-glands of the neck.

The affected half of the tongue was removed by operation together with the associated glands. On microscopic examination, the latter were found to be healthy.

## THYROGLOSSAL CYST OF TONGUE.



The tongue of a child divided by sagittal section.

In the mid-line of the base of the tongue is a small, oval cyst. It lies a short distance behind the circumvallate papillæ near the upper border of the body of the hyoid bone.

The cyst was originally filled with mucus.

*Hunterian Museum, R.C.S., 1206.1*

No clinical history.

#### XXXIV. MALIGNANT TUMOURS OF THE TONGUE AND MOUTH.

**Carcinoma** of the tongue is a common disease, and very much more common in men than in women. It is usually preceded by chronic superficial glossitis. Any part of the tongue may be affected, but the edges of the buccal as distinct from the pharyngeal portion are favourite sites owing to their liability to irritation.

Carcinoma commences as an ulcer, a warty growth, or a nodule in the mucous membrane. Whatever the form of the initial lesion, its most characteristic clinical peculiarity is induration. A carcinomatous ulcer has a hard, raised, and often everted edge, a rough and irregular floor, and a firm base which forms a plaque or mass incorporated with the substance of the tongue. The actual appearance of the surface of the ulcer and its liability to hæmorrhage are largely conditioned by the intensity of the secondary infection by bacteria present in the mouth.

In a section of the tongue carried through the growth, the latter appears as a white mass projecting down from the surface into the red of the muscle. Its edge may be sharply defined or may shade off through projecting lines and outlying nodules indicative of the general direction of infiltration.

There is a certain degree of constancy in the directions along which carcinoma spreads within the substance of the tongue. Thus, a growth commencing at one edge extends down along the hyoglossus to the hyoid bone and across the middle line into the opposite side. A carcinoma starting beneath the tip spreads back along the genioglossus in the base of the tongue. The portion of the tongue which is infiltrated by carcinoma loses its mobility, and ultimately the whole tongue becomes so fixed that it cannot be protruded from the mouth. Fixation of the tongue is also determined by extension of the growth beneath the mucous membrane of the floor of the mouth to the lower jaw. When the hyoglossus has been infiltrated down to the hyoid bone, the carcinoma may grow out in a continuous line round the posterior border of the mylohyoid into the submaxillary region of the neck.

Carcinoma of the tongue usually invades the lymphatic glands of the neck at an early stage, the glands involved being those of the submaxillary and jugular groups. Owing to the anatomical arrangement of the lymphatics of the tongue, there is a strong tendency to invasion of the glands on both sides of the neck. The submental glands are enlarged if the carcinoma involves the tip of the tongue.

The microscopic appearance is that of a squamous-cell carcinoma with cell-nests. From the growing edge on the surface branching and anastomosing columns of epithelial cells can be seen passing down among the muscle fibres. A round-celled infiltration of the connective tissue of the tongue often surrounds the edges of the growth.

**Sarcoma** of the tongue is rare.

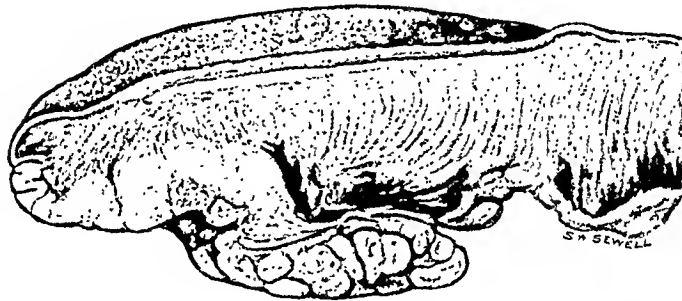
**Carcinoma of the Lip** (squamous-cell) is common, and is seen more often in the lower than in the upper lip and in men more often than in women. It commences as a warty growth or indurated crack and progresses rapidly to form a characteristic carcinomatous ulcer from the base of which a V-shaped

mass of white growth extends down into the lip. The submental and submaxillary glands are enlarged.

**Carcinoma of the Gum**, also of the squamous-cell variety, usually commences round an infected tooth, and appears on the surface as a warty growth or as an ulcer with raised, everted edges. It rapidly invades and destroys the jaw, and extends to the antrum if in the upper jaw, or outwards on to the skin surface in the case of the lower jaw. The associated lymphatic glands become enlarged.

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### CARCINOMA OF TONGUE.



One half of a tongue which has been divided by sagittal section.

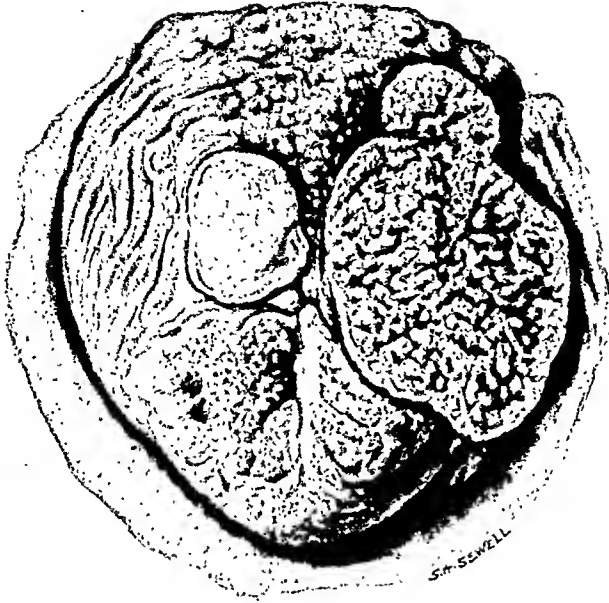
The under surface of the free part of the tongue is occupied by an ulcerated, white growth which has invaded the muscle of the tongue and the submaxillary gland.

*Hunterian Museum, R.C.S., 1796.2*

**MICROSCOPIC STRUCTURE.**—Squamous-cell carcinoma.

**CLINICAL HISTORY.**—The patient was a man, aged 65, who died of recurrence after operation for removal of the carcinoma of the tongue.

## PAPILLOMA AND CARCINOMA OF TONGUE.



A tongue drawn from the dorsal aspect.

Most of the papillæ have disappeared from the anterior two-thirds of the tongue and there are several fissures.

On the right half, close to the mid-line, there is a small, raised, ulcerated area, smooth on the surface and yellow in colour.

On the left half, extending from near the mid-line to the lateral border and on to the inferior surface, is a raised ulcer with a warty surface. It reaches from the circumvallate papillæ behind to within a short distance of the tip in front.

*Museum of the College of Medicine, University of Durham, Newcastle-upon-Tyne, 313/19*

**MICROSCOPIC STRUCTURE.**—Papilloma and squamous-cell carcinoma.

No clinical history.

## CARCINOMA OF TONGUE.

A horizontal section of a tongue below the dorsum.

The mucous surface of the tongue shows atrophy of the papillæ and areas of leucoplakia. The cut surface shows two deposits of carcinoma, one on each border.

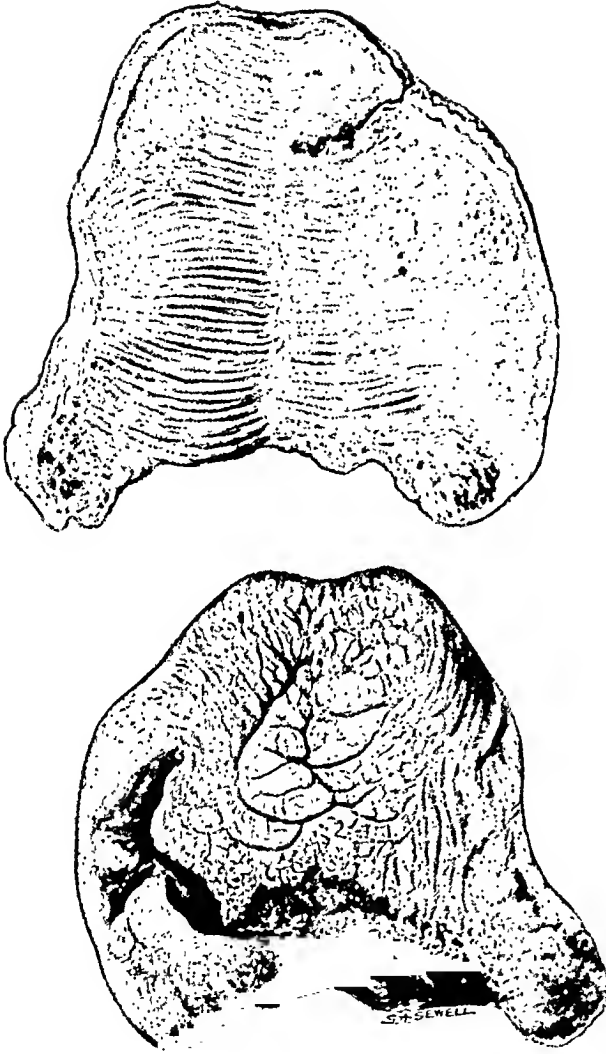
*Museum of University College Hospital, 15.A.G.18*

MICROSCOPIC STRUCTURE.—Squamous-cell carcinoma both sides.

CLINICAL HISTORY.—The patient was a man, aged 66, who had had a sore on the left side of his tongue for two months, but had not noticed any lump on the right side. He had syphilis at 19 years of age.

A bilateral gland operation was followed by removal of the tongue eleven days later. He died of recurrence in the floor of the mouth four months after operation.

# CARCINOMA OF TONGUE.



MUSEUM OF UNIVERSITY COLLEGE HOSPITAL 15.A.G.18



## CARCINOMA OF TONGUE.

A tongue drawn from the dorsal aspect and in section.

Over the anterior two-thirds of the dorsum the papillæ are atrophied and the mucous membrane is fissured.

The middle of the anterior part of the tongue is occupied by an ulcer with sinuous outlines. The posterior edge is smooth and rounded and shows a tendency to heal. The anterior edge is heaped up in a manner characteristic of a malignant ulcer.

The section shows infiltration of the muscle of the tongue by growth.  
*Museum of the College of Medicine, University of Durham, Newcastle-upon-Tyne, 313/7*

MICROSCOPIC STRUCTURE.—Squamous-cell carcinoma.

CLINICAL HISTORY.—It is stated that the carcinoma arose on a gummatous ulcer.

# CARCINOMA OF TONGUE.



MUSEUM OF THE COLLEGE OF MEDICINE, UNIVERSITY OF DURHAM, NEWCASTLE-UPON-TYNE, 312/7

## CARCINOMA OF TONGUE.

A tongue removed by operation at the level of the hyoid.

Over most of the anterior half the papillæ are atrophied. On the dorsum is a deep ulcer running longitudinally over the middle two-fourths. The floor of the ulcer is covered with sloughs and its margins are ragged and undermined. The neighbouring part of the tongue is pale and opaque owing to infiltration by growth, a nodule of which projects beneath the mucous membrane immediately behind the circumvallate papillæ to the medial side of the line of the ulcer. To the right of the main ulcer is a smaller one on the margin of the tongue.

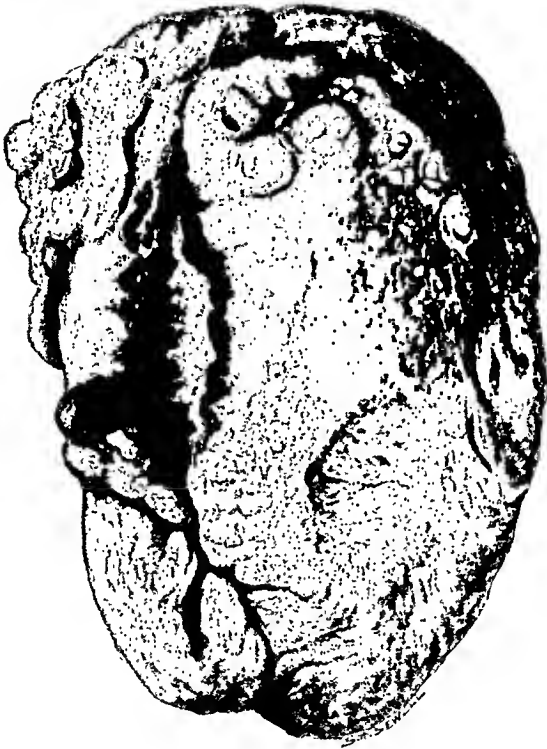
*Museum of University College Hospital, 15.A.G.3*

MICROSCOPIC STRUCTURE.—Squamous-cell carcinoma.

CLINICAL HISTORY.—The patient was a man, aged 65, who for ten years had suffered from recurrent ulceration of the tongue. Until two months before admission to hospital the ulcers had always healed. On admission his whole tongue was indurated, with a deep linear ulcer which bled readily. There were a few small enlarged glands on the right side of the neck. Wassermann reaction negative. He died four weeks after the operation.

AUTOPSY.—No local recurrence. Glands on the right side of the neck contained growth.

## CARCINOMA OF TONGUE.



MUSEUM OF UNIVERSITY COLLEGE HOSPITAL. 15.A.G.3

## CARCINOMA OF TONGUE.

One-half of a tongue divided by longitudinal section with the submaxillary lymphatic and salivary glands of the same side.

There is a malignant ulcer on the under surface of the tip of the tongue with infiltration extending backwards towards the hyoid bone. There are white deposits of growth in the glands.

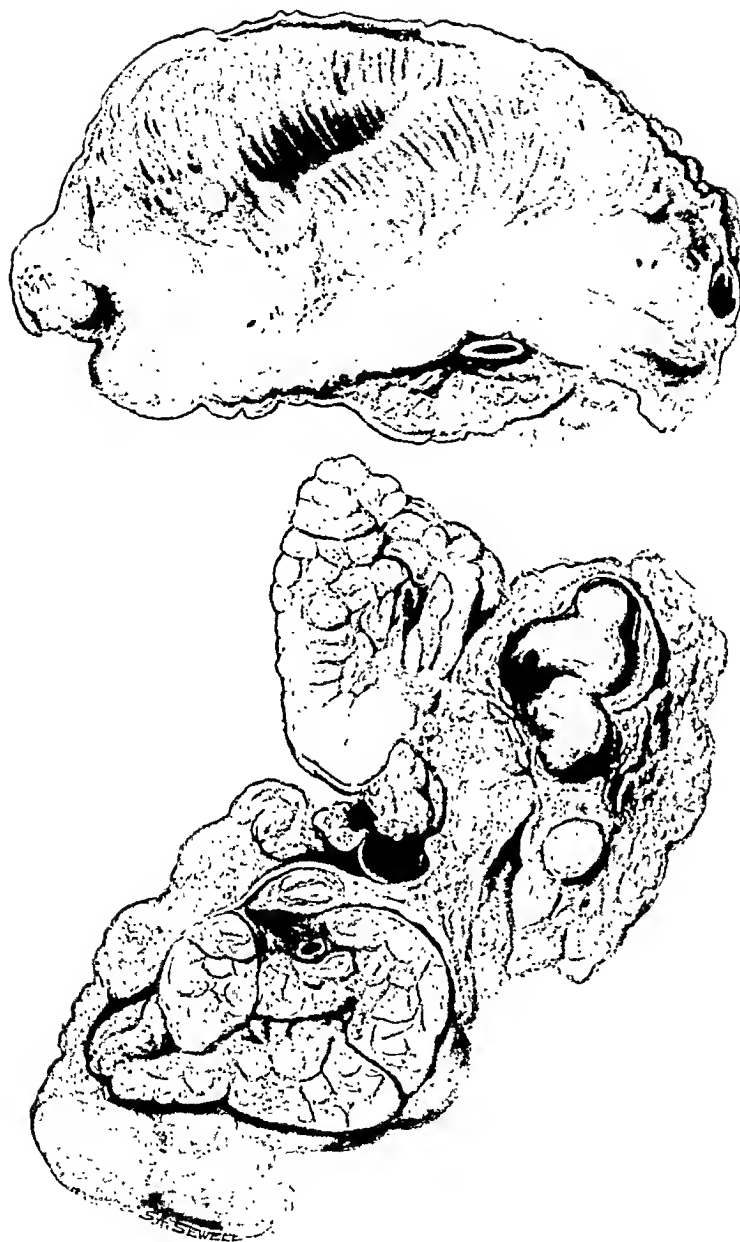
*Museum of University College Hospital, 15.A.G.10*

MICROSCOPIC STRUCTURE.—Squamous-cell carcinoma.

CLINICAL HISTORY.—The patient was a man, aged 51, who had had a painless lump in his tongue for three months. There was interference with speech but not with swallowing. His tongue was fixed in the floor of the mouth by an ulcerating growth along the anterior part of its right border. The growth came to the surface of the tongue at several other points and there were enlarged hard glands on both sides of the neck. He died three days after the removal of the tongue and glands.

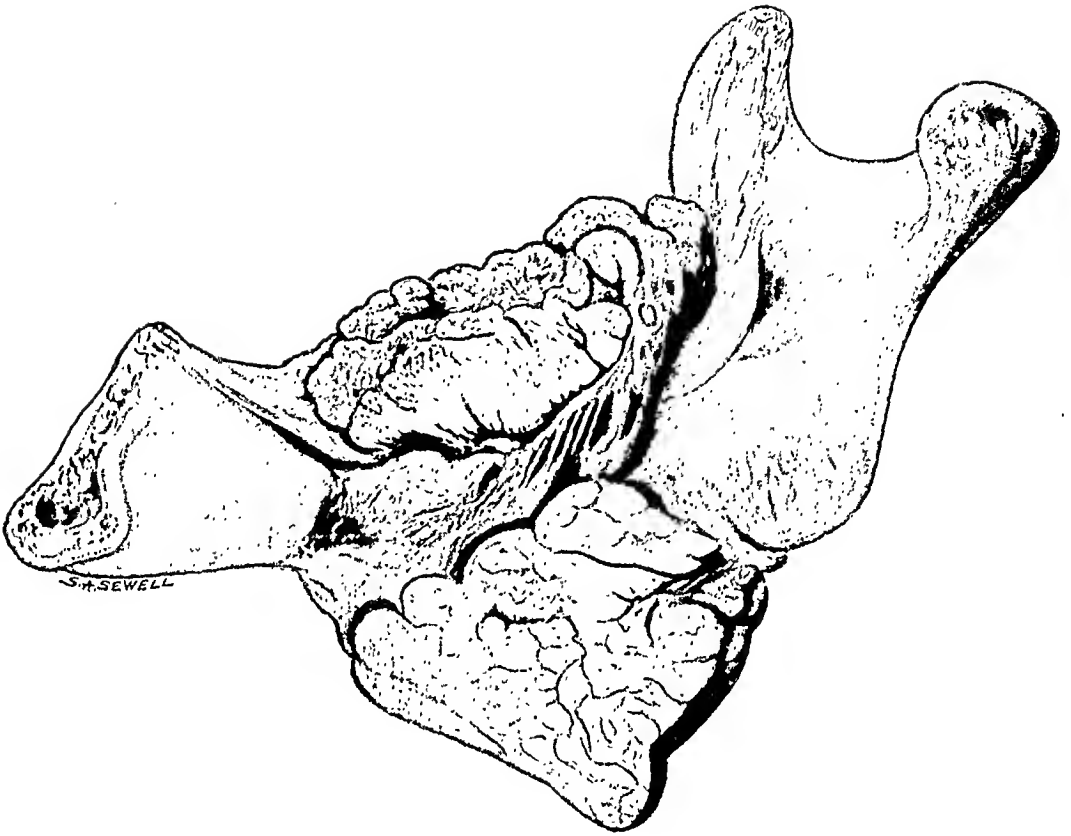
AUTOPSY.—Congestion of lungs.

# CARCINOMA OF TONGUE.



MUSEUM OF UNIVERSITY COLLEGE HOSPITAL, 15.A.G.10

## CARCINOMA OF GUM.



The right half of an edentulous mandible with a small portion of the cheek and the submaxillary salivary gland.

A coarsely papillary, ulcerated tumour has grown from the posterior part of the gum.

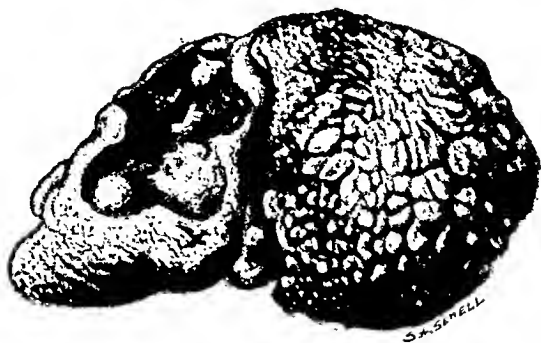
*Hunterian Museum, R.C.S., 1794.1*

**MICROSCOPIC STRUCTURE.**—Squamous-cell carcinoma. The submaxillary gland is free from growth.

**CLINICAL HISTORY.**—The patient was a man, aged 68, who had had a sloughing tumour within his mouth for three months. The growth involved the jaw and cheek.

The specimen illustrated was removed by operation. The patient died within twenty-four hours.

## PAPILLOMA AND CARCINOMA OF TONGUE.



The left half of a tongue.

There is an ulcerated growth on the anterior part of the tongue with a papilloma behind.

*Museum of King's College Hospital, R.11*

**MICROSCOPIC STRUCTURE.**—Squamous-cell carcinoma with cell-nests and papilloma.

**CLINICAL HISTORY.**—The patient was a man, aged 60. The specimen illustrated was removed by operation. The immediate result was satisfactory. No after-history.



## CARCINOMA OF FLOOR OF MOUTH.



Anterior one-third of a tongue with the mucous membrane of the floor of the mouth and the alveolar margin of the mandible.

A shallow ulcer with raised edges occupies the middle line between the under surface of the tip of the tongue and the alveolar margin.

*Museum of King's College Hospital, R.14*

### MICROSCOPIC STRUCTURE.—Epithelioma.

CLINICAL HISTORY.—The patient was a man, aged 51, who had had a sore beneath the tip of his tongue for two months. It commenced as a small hard lump which gradually increased in size and ulcerated. The specimen was removed by operation.

AFTER-HISTORY.—The patient was alive and well four years after operation.

## CARCINOMA OF FLOOR OF MOUTH.



The left alveolar margin and part of the right side of a mandible with the left half of the floor of the mouth and the left half of the anterior two-thirds of the tongue.

An extensive ulcer occupies the floor of the mouth and the border of the tongue. Its edges are white, raised, and hard.

*Museum of King's College Hospital, R.13*

**MICROSCOPIC STRUCTURE.**—Squamous-cell carcinoma with cell-nests.

**CLINICAL HISTORY.**—The patient was a man who for two months had suffered pain in the left side of his tongue and had noticed an ulcer which he thought was due to a carious molar. The tongue was fixed. There were no enlarged glands. The specimen was removed by operation.

**AFTER-HISTORY.**—Death two years after operation, cause unknown.

## CARCINOMA OF LIP.

The lower lip and the left extremity of the upper lip with its surrounding skin.

The whole length of the lower lip and the left angle of the mouth is occupied by an irregular ulcer with a nodular surface and raised everted edge.

*Museum of King's College Hospital, R34A*

MICROSCOPIC STRUCTURE.—Epithelioma.

CLINICAL HISTORY.—The patient was a man, aged 65, who for three and a half years had had an ulcer on his lower lip which gradually spread round the left corner of the mouth. He was a farmer and a heavy pipe smoker. On examination the growth was hard and infiltrated the tissues of the lower lip. There were enlarged hard glands in both submaxillary triangles. The specimen was removed by operation with the affected glands, and the patient made an uneventful recovery.

AFTER-HISTORY.—Death from recurrence five months after operation.

# CARCINOMA OF LIP.



MUSEUM OF KING'S COLLEGE HOSPITAL, R.34.A  
NO. 27—SUPPLEMENT

G 1

### XXXV. DIVERTICULUM OF THE PHARYNX.

A DIVERTICULUM is a pouch which arises from the middle of the posterior wall of the pharynx close to its junction with the œsophagus. It consists of mucous membrane and submucosa over which is spread a partial covering of muscle fibres derived from the inferior constrictor. It projects backwards between the horizontal lower fibres and the oblique fibres of which the main part of the muscle is composed.

It is possible that a congenital weakness of the pharyngeal wall at this point may be a factor in the causation of the diverticulum, and it has been suggested that the pressure of food backwards from the rigid posterior surface of the cricoid cartilage is a determining factor in its development.

Diverticulum of the pharynx is more common in men than in women. The symptoms of dysphagia, which appear for the first time in middle life, are due to the fact that the mouth of the diverticulum is wider and more directly in line with the axis of the pharynx than is the upper aperture of the œsophagus. Food therefore passes into and distends the diverticulum, which, when full, occupies the space between the vertebral column and the œsophagus and compresses the latter against the relatively rigid trachea. As the pouch enlarges it is usually displaced to the left of the middle line so as to form a swelling in the neck from which unaltered food can be squeezed back into the mouth.

Diverticulum of the pharynx is occasionally associated with stenosis of the upper end of the œsophagus.

## DIVERTICULUM OF PHARYNX.

The viscera of the neck in sagittal section.

The diverticulum projects from the posterior wall of the pharynx opposite the cricoid cartilage. Its wall is composed for the most part of fibrous tissue lined by mucous membrane, but the muscular tissue of the pharyngeal wall can be traced for about  $\frac{3}{4}$  in. from its mouth. From the side the diverticulum is seen to be in contact with the posterior border of the thyroid gland. From above, the mouth of the diverticulum appears as a direct continuation of the cavity of the pharynx and entirely obscures the upper end of the œsophagus. The reason for the passage of food into the pouch rather than into the œsophagus is clear when the specimen is examined from this aspect. The upper end of the œsophagus is compressed by the diverticulum, but is not narrowed by any structural alteration of its walls.



FROM ABOVE.

*Hunterian Museum, R.C.S., 6031.1*

**CLINICAL HISTORY.**—The patient was a man, aged 75, who had had difficulty in swallowing, with vomiting of unaltered food, for six months before admission to hospital. He was able to swallow both solids and fluids if they were taken slowly. He “brought up wind when he shaved the left side of his neck”.

X-rays showed the pouch projecting from the posterior wall of the pharynx and extending down to the level of the top of the sternum.

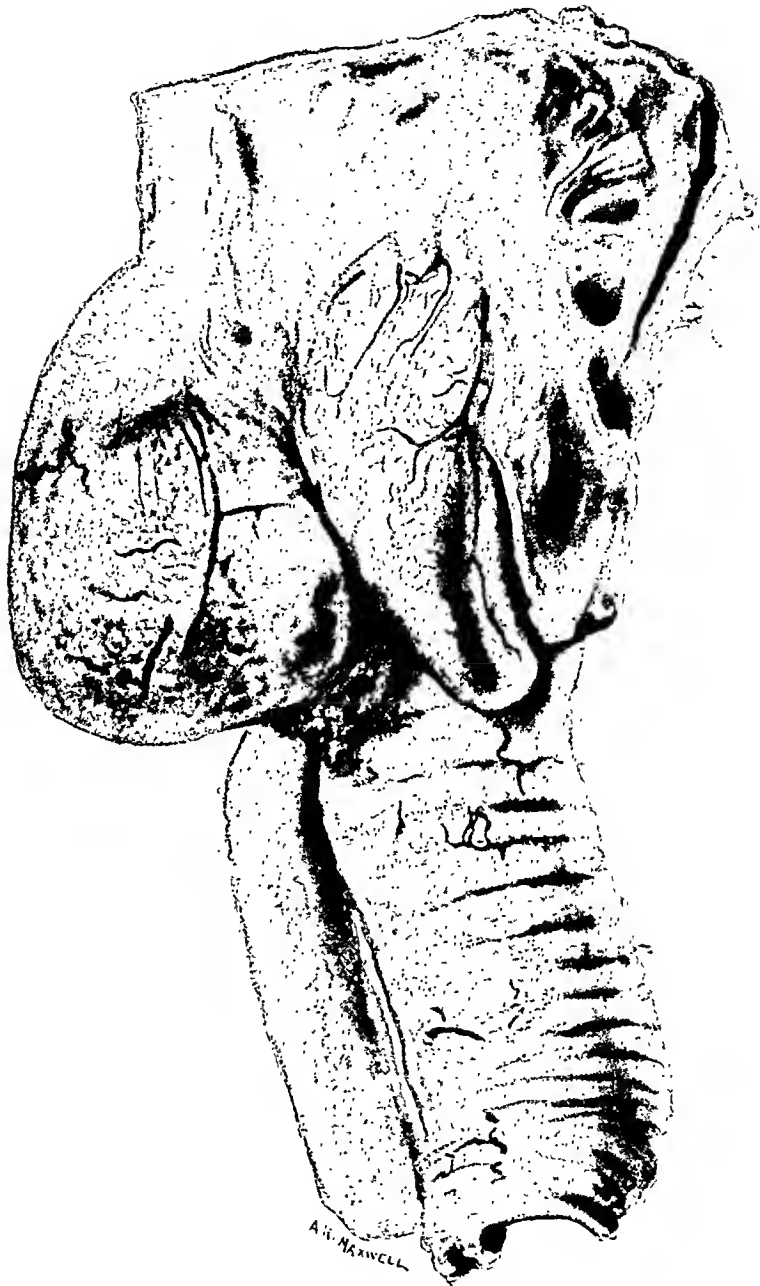
Direct examination showed the opening of the pouch and irregular ulceration of the posterior part of the left vocal cord.

Wassermann reaction negative.

Death from bronchitis nineteen days after admission.

**AUTOPSY.**—Papillomata at posterior ends of the vocal cords and arytenoids.

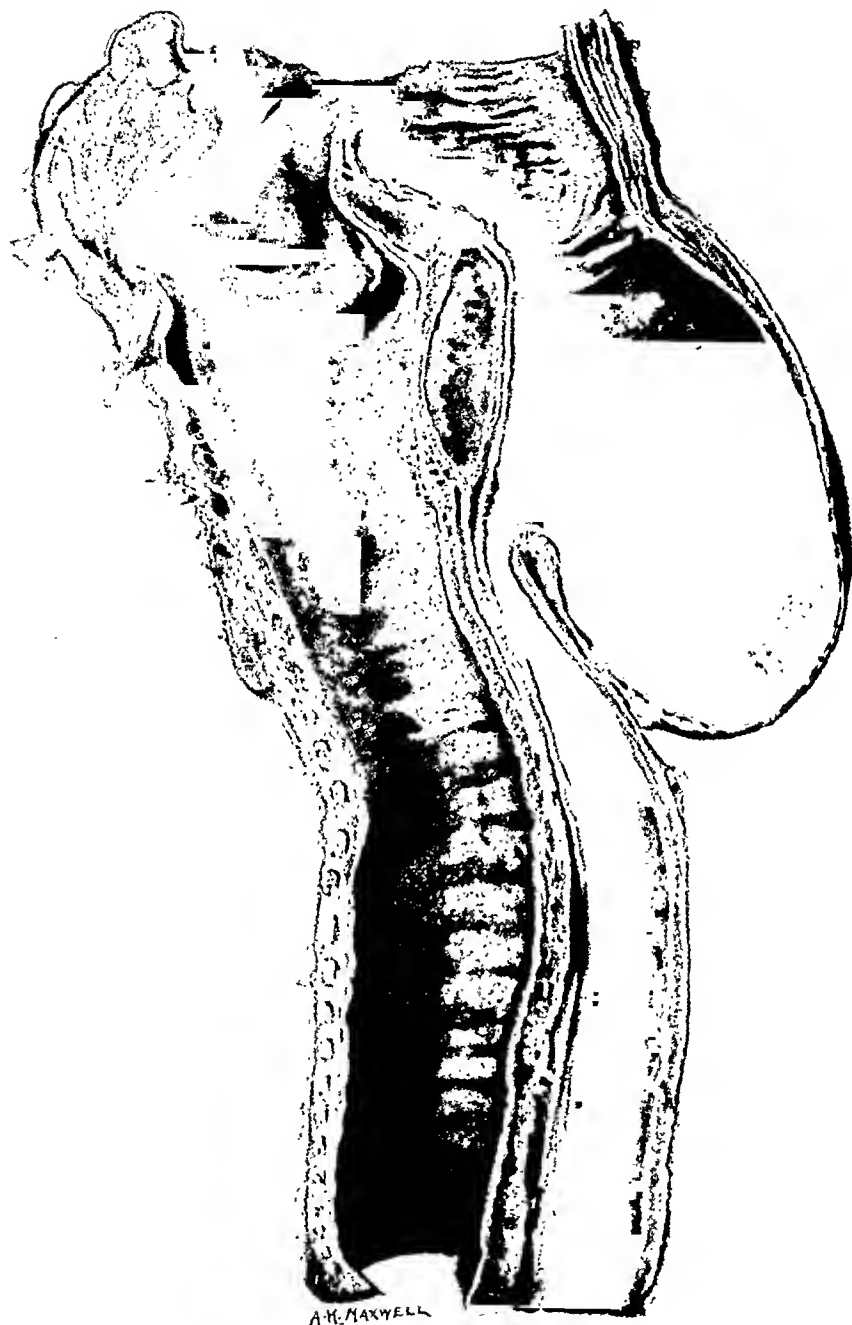
DIVERTICULUM OF PHARYNX.



FROM OUTSIDE.

HUNTERIAN MUSEUM, R.C.S. 6031.1

DIVERTICULUM OF PHARYNX.



SECTION.

HUNTERIAN MUSEUM, R.C.S., 6031.1



## DIVERTICULUM OF PHARYNX.

Parts of a pharynx and œsophagus with the larynx and the upper portion of the trachea.

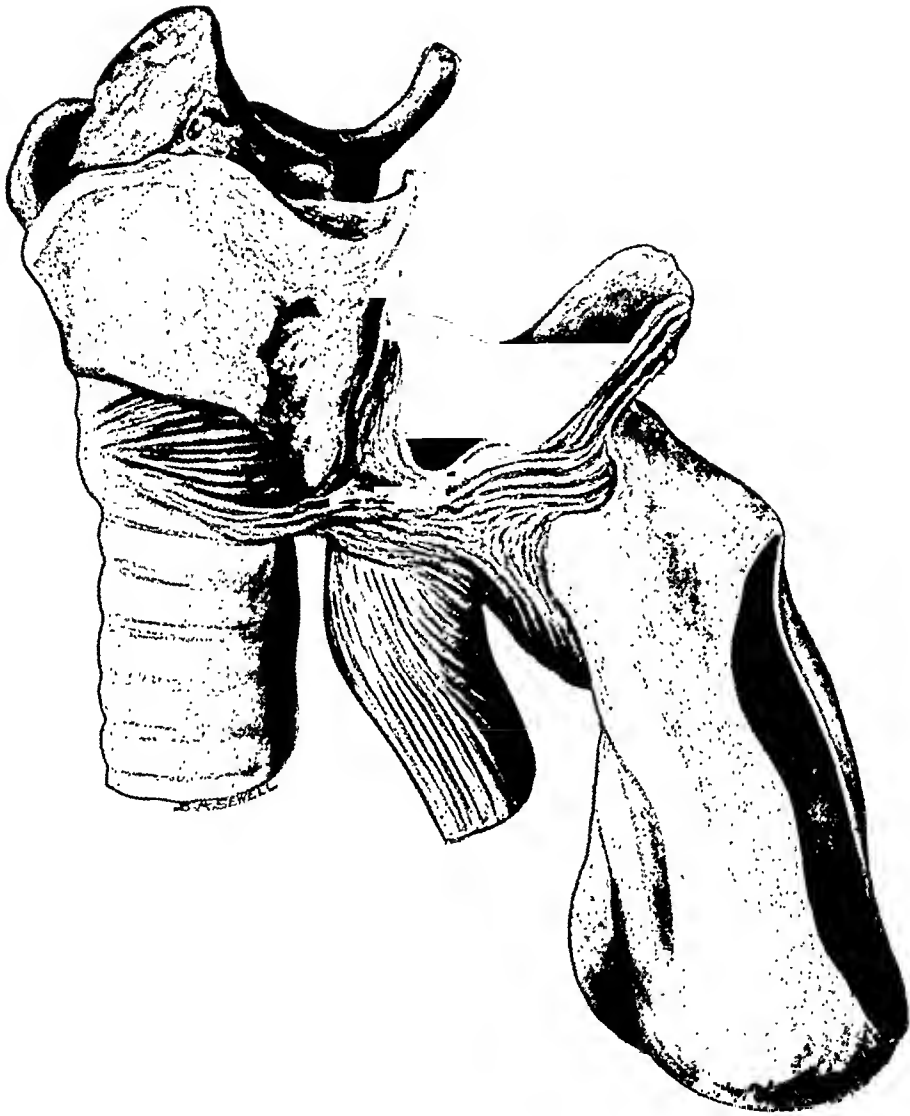
A pear-shaped diverticulum  $\frac{1}{4}$  in. long and 2 in. wide projects from the posterior surface of the lower end of the pharynx. It communicates with the pharynx by an orifice 1 in. in diameter situated immediately above the transverse fibres of the inferior constrictor muscle.

*Hunterian Museum, R.C.S., 6032.1*

**MICROSCOPIC STRUCTURE.**—The wall of the diverticulum is composed of mucous membrane, vascular connective tissue, and scattered bundles of muscle.

**CLINICAL HISTORY.**—The patient was a man, aged 49, who had suffered from progressive dysphagia and vomiting for ten years. Attempts to pass a bougie failed, the instrument being arrested 7 in. from the teeth. He became very emaciated, and eventually died three days after a gastrostomy.

# DIVERTICULUM OF PHARYNX.



HUNTERIAN MUSEUM, R.C.S., 6032.1

## XXXVI. MALIGNANT TUMOURS OF THE PHARYNX.

MALIGNANT tumours of the pharynx may be classified according to their origin in the nasopharynx, oral pharynx, or hypopharynx (laryngeal pharynx).

In the *nasopharynx* carcinoma is a rare disease. Its usual site is the lateral wall, where it is apt to invade the Eustachian tube, soft palate, and the branches of the second division of the maxillary nerve in the sphenomaxillary fossa. Extra-pharyngeal tumours arising from the base of the skull may involve the pharynx secondarily. The special tumour of this type is a fibroma which grows from the periosteum of the basi-occipital and distends the nose and upper part of the face. It is a disease of young adult males. Very rarely a teratoma arising from the anterior growing point of the embryo occurs as a congenital growth in this situation.

Tumours of the *oral pharynx* arise chiefly in the tonsil or its immediate neighbourhood. Apart from carcinoma, the chief types are endothelioma and lymphosarcoma.

Tumours of the *hypopharynx* include three main groups:—

1. Those which arise around the upper aperture of the larynx—epilaryngeal—and interfere with the upper aperture at an early stage of their development.

2. Tumours of the sinus pyriformis, which often reach an advanced stage before causing symptoms. The main direction of extrapharyngeal extension of a carcinoma of the sinus pyriformis is outwards through the ala of the thyroid cartilage and to the glands of the deep jugular chain. Carcinoma in these two sites is almost confined to men.

3. Post-cricoid carcinoma, which is a disease of women, and arises at an age earlier than that usually associated with carcinoma. It commences on the pharyngeal mucous membrane covering the back of the cricoid cartilage, and spreads in a tubular form around the lowest part of the pharynx, producing a gradually increasing stenosis.

In addition to these relatively common types are the carcinoma of the lateral pharyngeal wall, which is a disease of males, and the carcinoma arising in the posterior laryngeal wall, which is more common in men but also occurs in women.

All forms of carcinoma of the hypopharynx are of squamous epithelioma type.

## CARCINOMA OF HYPOPHARYNX.

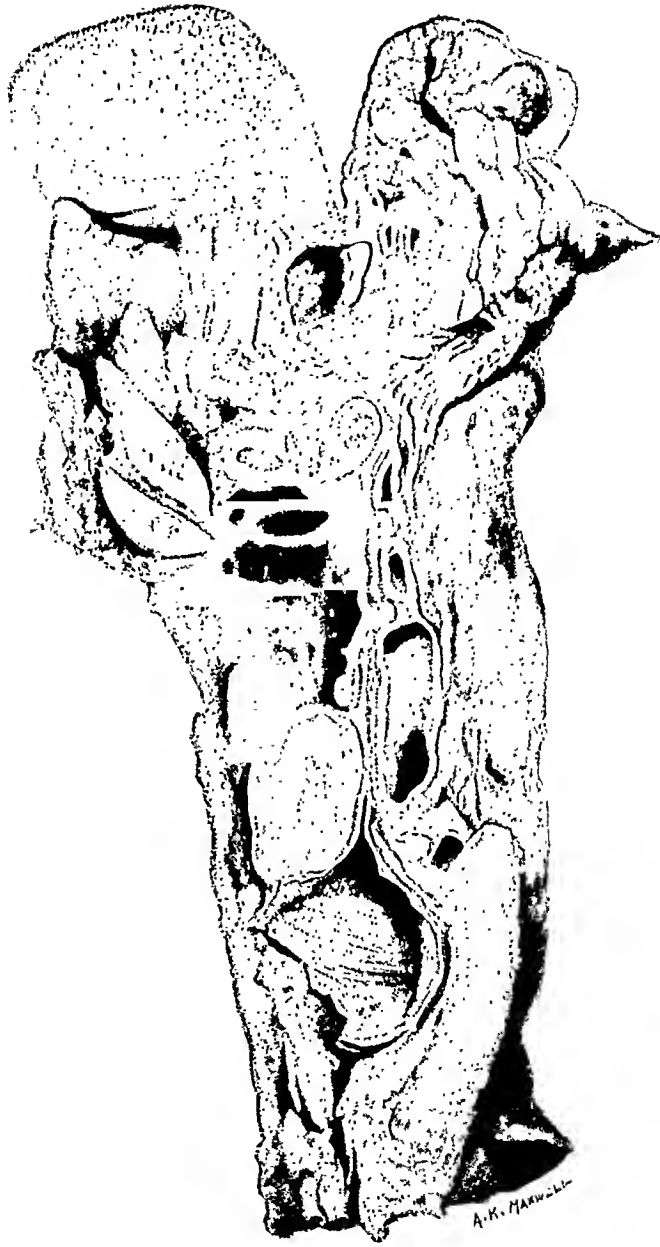
The viscera of a neck in longitudinal section.

The wall of the pharynx and upper part of the œsophagus is infiltrated by a white growth for a length of 6 cm. The growth is cylindrical in shape and has almost obliterated the lumen of the pharynx. In front it extends on to the back of the arytenoids and causes fixation of the vocal cords by limiting the movement of the crico-arytenoid joint. At a lower level it infiltrates the cricoid cartilage, and a nodule projects forwards beneath the mucous membrane of the trachea.

*Museum of University College Hospital, 13.AH.3*

CLINICAL HISTORY.—The patient was a woman, aged 57, who was admitted to hospital in a state of starvation. For six years she had had hoarseness and occasional loss of voice. For eight months she had been losing weight. For five months she had had difficulty in swallowing. The primary growth could not be felt through the neck, but there was a hard palpable gland. She died a few days after gastrostomy had been performed. The gland in the neck contained growth.

# CARCINOMA OF HYPOPHARYNX.



FROM OUTSIDE. -

MUSEUM OF UNIVERSITY COLLEGE HOSPITAL, 13.A.H.3

CARCINOMA OF HYPOPHARYNX.



SECTION.

MUSEUM OF UNIVERSITY COLLEGE HOSPITAL, 13.AH.3

## CARCINOMA OF SINUS PYRIFORMIS.

A pharynx, larynx, and base of tongue. The pharynx has been opened from behind.

The right pyriform sinus is occupied by an ulcerating growth with raised, irregular margins. The growth has invaded the right ary-epiglottic fold and has extended across the epiglottis to the opposite side.

On the right side of the specimen is an enlarged lymph gland containing growth.

*Hunterian Museum, R.C.S., 1799.1*

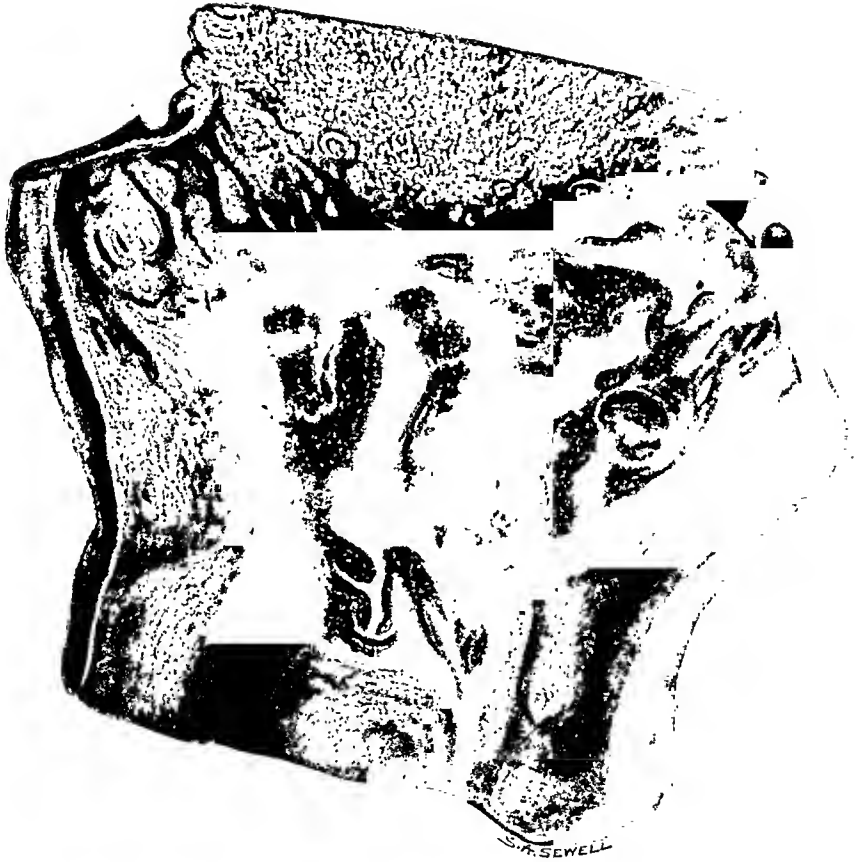
MICROSCOPIC STRUCTURE.—Squamous-cell epithelioma.

CLINICAL HISTORY.—The patient was a man, aged 64, who complained of a sore throat for a year. For three months he had had a continuous burning pain in his throat, and for one month his voice had been husky. He was spitting blood and mucus.

On examination there were hard, enlarged glands on both sides of the neck, and, in the position of the epiglottis, an irregular mass which bled when touched. Tracheotomy was performed two weeks before death.

AUTOPSY.—There was a secondary growth in the lower part of the left lung continuous with a mass in the bronchial glands. There was an abscess in the base of the right lung.

CARCINOMA OF SINUS PYRIFORMIS.



HUNTERIAN MUSEUM, R.C.S., 1799.1



## CARCINOMA OF ARY-EPIGLOTTIC FOLD.

A pharynx with the tongue and larynx. The pharynx has been opened from behind.

An ulcerated carcinoma has grown from the back of the right ary-epiglottic fold. It fills the right pyriform sinus and extends round the posterior wall of the pharynx to the left side. On the right side the growth has extended out into the glands of the neck, displacing the common carotid artery outwards and backwards. The mucous membrane lining the left pyriform sinus is intact.

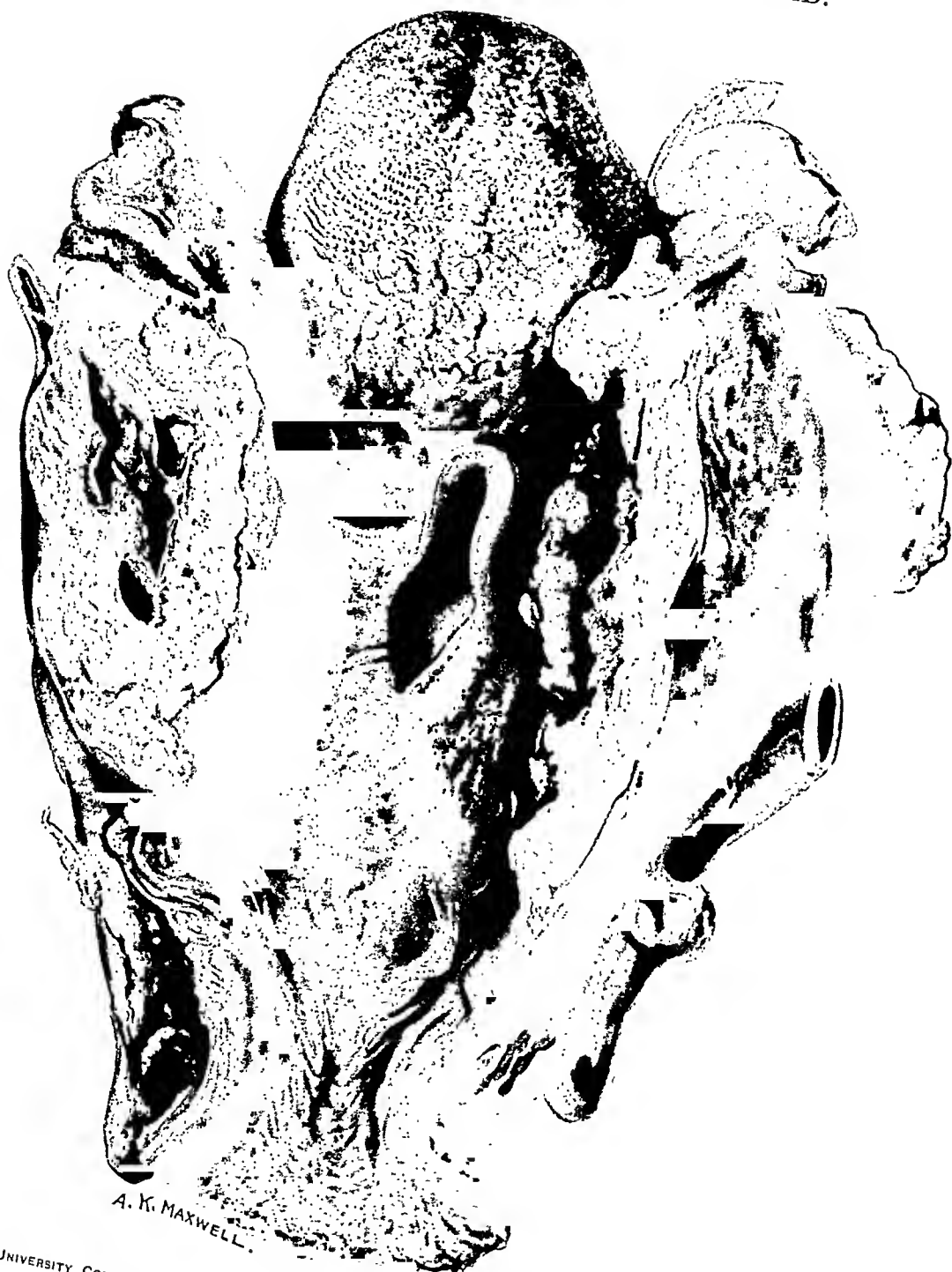
*Museum of University College Hospital, 12.AH.4*

MICROSCOPIC STRUCTURE.—Squamous epithelioma.

CLINICAL HISTORY.—The patient was a man, aged 59, who was admitted to hospital with urgent dyspnœa, for which tracheotomy was performed under local anæsthesia. He died nine days later.

AUTOPSY.—Œdema of lungs.

CARCINOMA OF ARY-EPIGLOTTIC FOLD.



A. K. MAXWELL.

MUSEUM OF UNIVERSITY COLLEGE HOSPITAL, 12.AH.4

## SARCOMA OF PHARYNX.

A pharynx with the larynx and base of the tongue, opened from behind.

Four tumours have grown beneath the mucous membrane. The largest is oval in outline and involves the left ary-epiglottic fold. A second lies in front of the epiglottis and is continued forwards over the base of the tongue to the left tonsil. The right tonsil is greatly enlarged, and in contact with it is a fourth tumour projecting backwards from the base of the tongue. It is probable that the enlarged right tonsil represents the primary tumour to which the others are secondary.

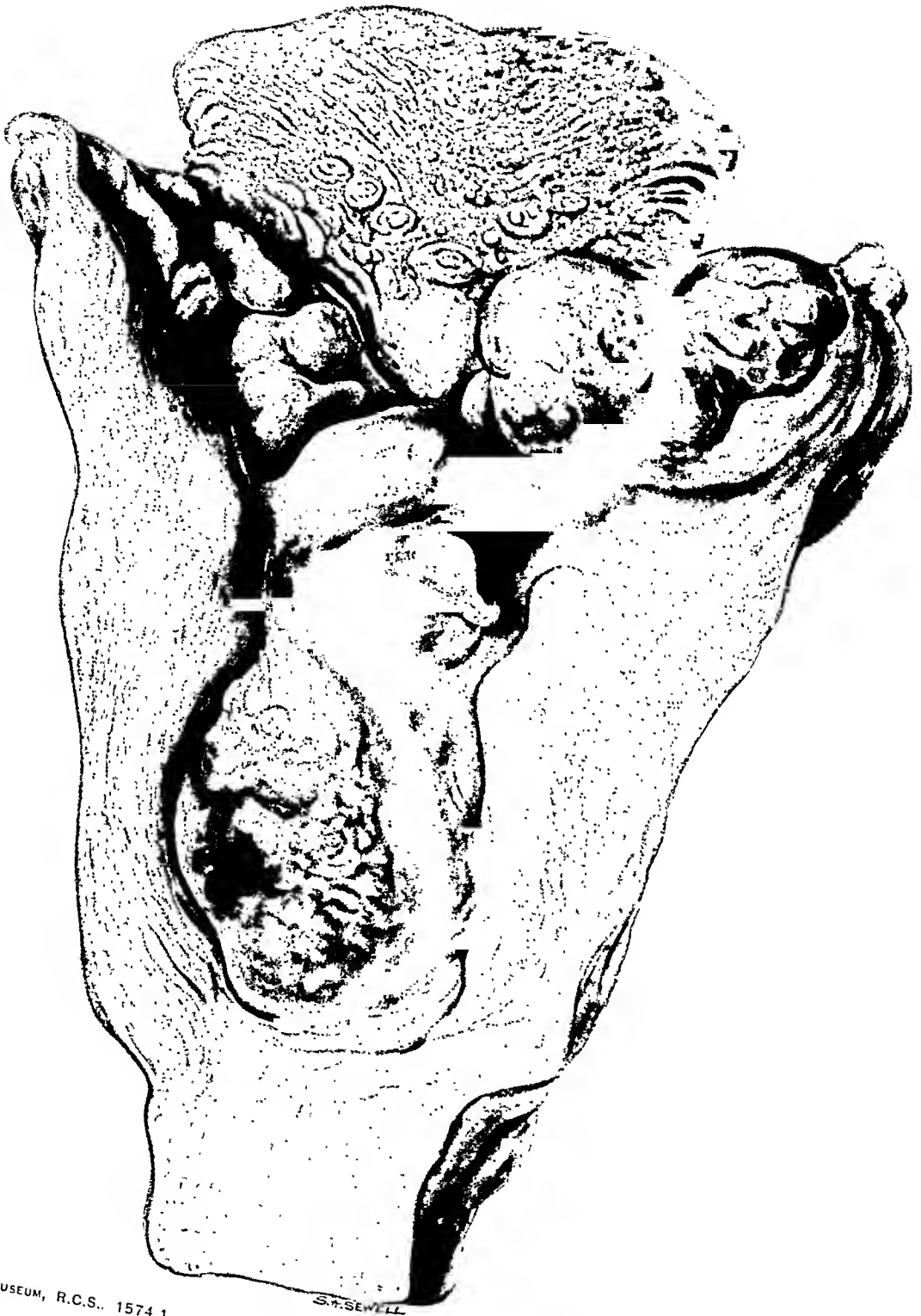
*Hunterian Museum. R.C.S., 1574.1*

MICROSCOPIC STRUCTURE.—Lymphosarcoma.

CLINICAL HISTORY.—The patient was a man, aged 71, in whom the tumours began to grow eighteen months before admission to hospital. Eventually respiration became so difficult as to necessitate tracheotomy. There were many enlarged, soft glands in the neck.

AUTOPSY.—The mucous membrane of the stomach was thickened by infiltration with small round cells similar to those which composed the tumours of the pharynx. The mesenteric glands were slightly enlarged.

SARCOMA OF PHARYNX.



HUNTERIAN MUSEUM, R.C.S.. 1574.1

## XXXVII. DIVERTICULUM OF THE ŒSOPHAGUS.

(*Traction Diverticulum.*)

A SMALL diverticulum from the middle or lower part of the œsophagus may be produced by the shrinking of some inflammatory focus, such as a tuberculous gland, which is adherent to its outer surface. The gland becomes apposed to the œsophagus when it is in a state of inflammatory swelling. When the tuberculous process subsides as the result of fibrosis the gland shrinks and approximates the two structures to which its poles are adherent. One pole of the gland is usually attached to the bifurcation of the trachea, and hence the diverticulum is drawn up from the œsophagus so that its axis is either horizontal or slopes downwards towards the mouth. For this reason it rarely happens that food collects within it to cause pressure or inflammation, and the existence of the diverticulum is seldom suspected during life. Occasionally the mouth of the diverticulum is directed upwards, and the symptoms then resemble those of the more common diverticulum of the pharynx.

### DIVERTICULUM OF ŒSOPHAGUS.

An œsophagus with the larynx, trachea, and part of the right lung.

Immediately below the bifurcation of the trachea is a diverticulum arising from the right anterior aspect of the œsophagus. It is hemispherical in shape, with a diameter of  $1\frac{1}{2}$  in., and its apex is attached to a tuberculous gland below the bifurcation of the trachea. The rest of the œsophagus is normal.

At the apex of the right lung is an irregular cavity surrounded by dense fibrous tissue. In the lower part of the lung are scattered miliary tubercles.

*Hunterian Museum, R.C.S., 6077.2*

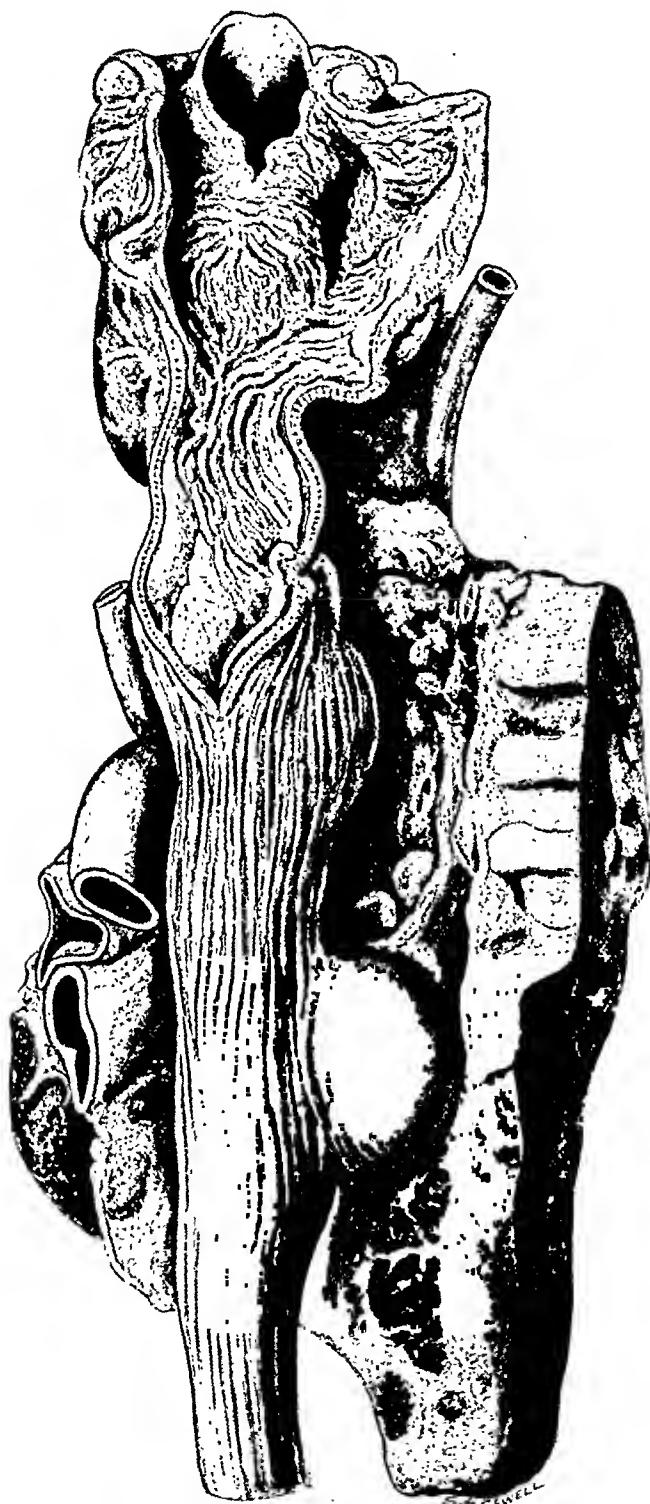
MICROSCOPIC STRUCTURE.—The wall of the diverticulum contains representatives of all the layers of the œsophageal wall. The lung is tuberculous.

CLINICAL HISTORY.—The patient was a man, aged 52, who suffered from chronic pulmonary tuberculosis. For nine months before his death he had complained of indigestion with a feeling of fullness after taking solid food. Towards the end he felt that there was a lump behind the sternum where food was arrested. He often vomited a quarter of an hour after meals and this gave him relief. The eating of solids was difficult and was immediately followed by regurgitation.

The pouch was seen by X rays.

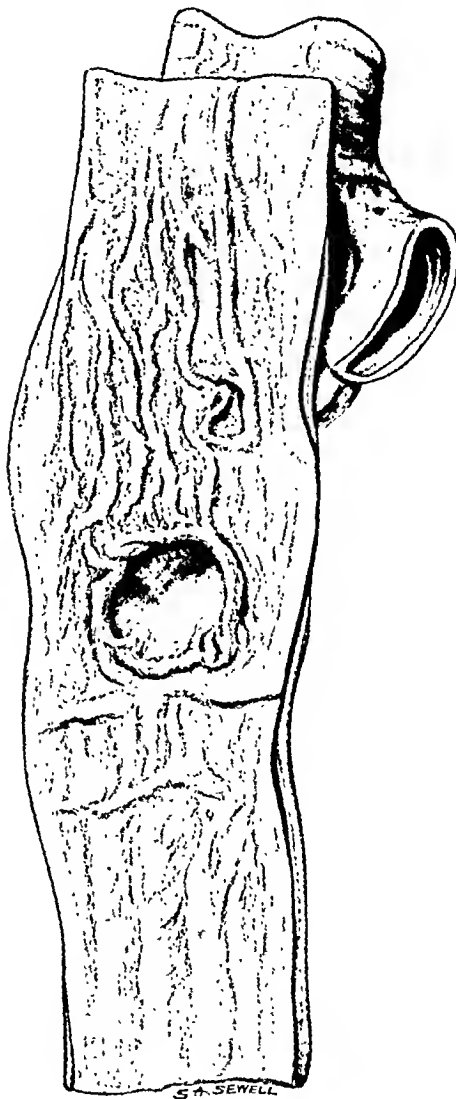
Death from exhaustion.

DIVERTICULUM OF ŒSOPHAGUS.



HUNTERIAN MUSEUM, R.C.S. 6077.2

## DIVERTICULUM OF ŒSOPHAGUS.



Part of an œsophagus with the bifurcation of the trachea.

Immediately below the bifurcation is a small traction diverticulum of the œsophagus the size of a pea. A short distance lower down is a second pouch  $\frac{2}{3}$  in. in diameter. The pouches protrude through the separated muscular fibres of the œsophagus, and each is adherent to a pigmented lymph gland.

*Hunterian Museum, R.C.S., 6076.1*

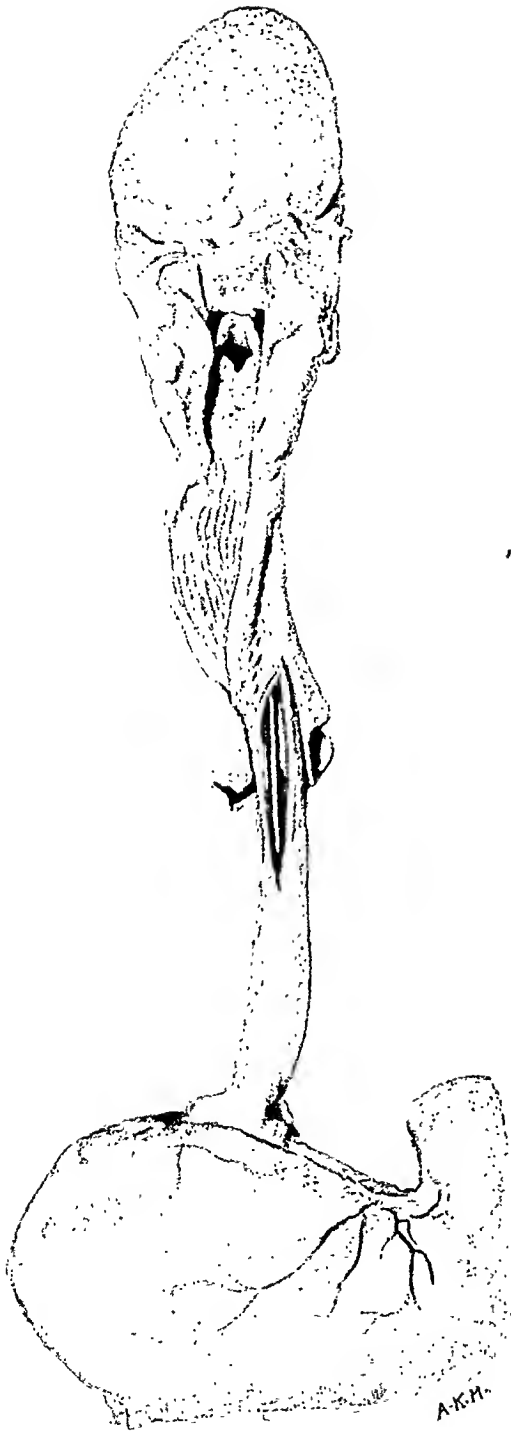
CLINICAL HISTORY.—From a man of 72.

### XXXVIII. CONGENITAL MALFORMATIONS OF THE ŒSOPHAGUS.

THE trachea and œsophagus are developed from the foregut and are differentiated from one another by the fusion of the two lateral halves of the œsophago-tracheal septum. These fuse last at the caudal end, and at this point an œsophago-tracheal fistula may be left. The upper part of the œsophagus usually ends blindly opposite the fistula.

The congenital abnormalities compatible with life are stricture and a thoracic position of the cardia. In the latter condition, a cone of stomach passes up through the diaphragm to join the œsophagus about the level of the 8th dorsal vertebra. There may be a stricture at the junction.





## CONGENITAL ATRESIA OF ŒSOPHAGUS.

(TRACHEO-ŒSOPHAGEAL FISTULA.)

The tongue, larynx, œsophagus, trachea, and stomach of an infant.

The upper part of the œsophagus is dilated and ends blindly opposite the lower part of the trachea. The lower part of the œsophagus opens into the trachea a short distance above the bifurcation. On the anterior aspect of the stomach is a gastrostomy opening. Gastrostomy was performed five days after birth on account of inability of the child to take food. Death one week later.

*Museum of University College Hospital,*  
1R2

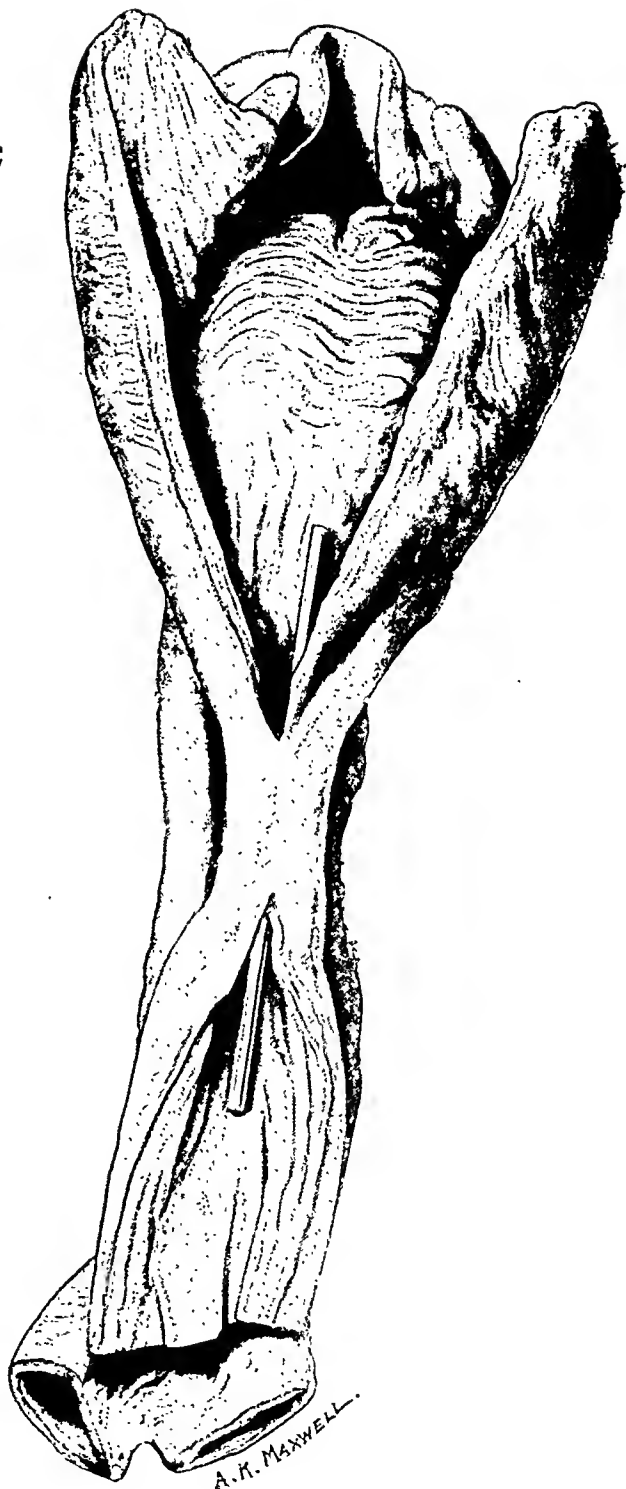
## CONGENITAL STRICTURE OF ŒSOPHAGUS.

A larynx and trachea with the lower part of the pharynx and corresponding portion of the œsophagus.

One inch below the lower border of the cricoid cartilage the lumen of the œsophagus is reduced in size so as to admit only a No. 6 catheter. Both the mucous membrane and the muscular tissue of the œsophagus at the site of stricture, as well as above and below it, are healthy. The inferior constrictor of the pharynx is hypertrophied. The upper cornua of the thyroid cartilage are relatively near to one another. From the absence of scarring it is probable that the stricture is of congenital origin.

*Museum of University College  
Hospital, 1R3*

**CLINICAL HISTORY.** — The patient was a man of between 70 and 80 years of age who had had symptoms of a stricture of the œsophagus for many years.



### XXXIX. CARDIO-SPASM.

(*Achalasia ; Œsophagectasia.*)

IN this condition there is imperfect co-ordination of the neuromuscular mechanism of the œsophagus in the sense that the cardiac sphincter is either excited to spasm or fails to relax during the act of swallowing. The clinical manifestation of this is dysphagia, and the anatomical result is progressive dilatation and hypertrophy of the lower three-fourths of the œsophagus.

The disease begins between the ages of 20 and 40 and is twice as common in women as in men. The subjects are frequently of a nervous type, and have occasionally been found to have a chronic ulcer of the stomach or duodenum.

The dilated portion of the œsophagus has its muscular coat increased in thickness and its mucous membrane congested and frequently ulcerated. The muscular hypertrophy stops a short distance above the cardia, which is normal in appearance and affords no organic cause of obstruction. When examined at operation, a finger passed up through the stomach is tightly gripped by the cardiac sphincter.

The dilatation of the œsophagus is progressive, and according to the stage at which it is examined the viscus may be fusiform or pear-shaped, or show an increased length, which is accommodated by a sigmoid curve above the diaphragm. The lowest inch or more of the œsophagus from the upper limit of the cardiac sphincter to the junction with the stomach remains of normal calibre.

## CARDIO-SPASM.

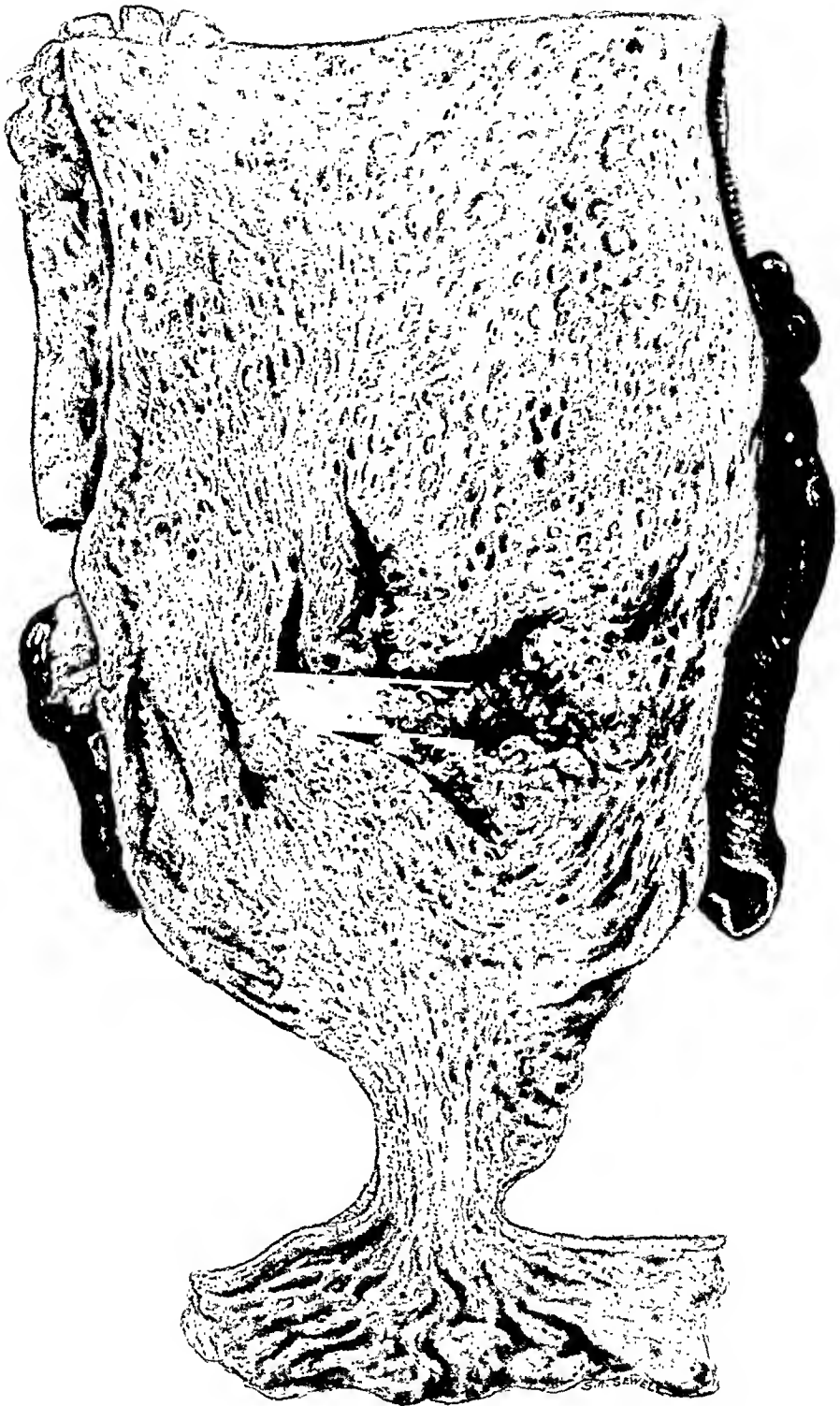
The lower part of an œsophagus opened out with a portion of the cardiac end of the stomach, parts of the right lung, trachea, and aorta. The œsophagus has been cut across about  $1\frac{1}{2}$  in. above the bifurcation of the trachea.

The œsophagus is greatly dilated and measures  $5\frac{1}{2}$  in. in circumference. At about 2 in. from the lower end it suddenly contracts, and at the cardia has a circumference of 1 in. only. Except at the lowest part the wall is thickened by hypertrophy of its muscular coat and by chronic inflammation of its mucous membrane. Four inches above the lower end there is localized bulging, 3 in. in diameter, of the anterior wall. For the most part the surface of the mucous membrane is covered with small, shallow, acute ulcers. There is no scarring round the cardia. The glands along the œsophagus are not enlarged. The glands in the hilum of the lung are enlarged and caseous, but are not adherent to the œsophagus. The lower lobe of the lung is consolidated and contains numerous miliary tubercles. The upper lobe has collapsed.

*Hunterian Museum, R.C.S., 6075.1*

CLINICAL HISTORY.—The patient was a man, aged 32, who complained of wasting and indigestion which had commenced at the age of 14 and was associated with vomiting immediately or shortly after food. Ten months before he was admitted to hospital he had a severe hæmatemesis lasting for four days. He became emaciated and died of broncho-pneumonia.

CARDIO-SPASM.



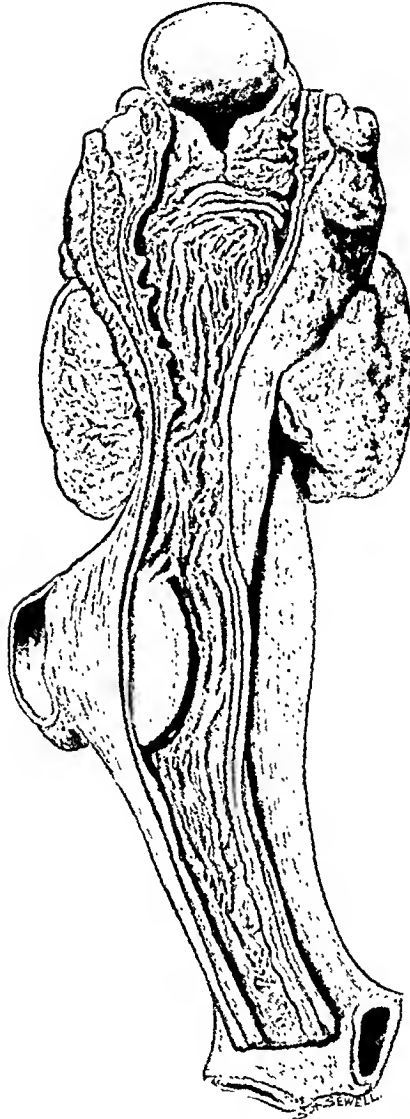
HUNTERIAN MUSEUM, R.C.S., 6075.1

## **XL. SIMPLE TUMOURS OF THE ŒSOPHAGUS.**

**S**IMPLE tumours of the œsophagus cause no symptoms unless and until they become polypoid. They are most often found towards the upper part of the œsophagus in old men. Fibromyoma, fibroma, and lipoma are the usual varieties. All are rare.

[**Œsophageal Varix.**—This condition is illustrated in the **ATLAS OF PATHOLOGICAL ANATOMY**, Part 8, April, 1927, p. 177.]

## BRANCHIAL CYST.



The viscera of a neck with the pharynx and œsophagus opened from behind.

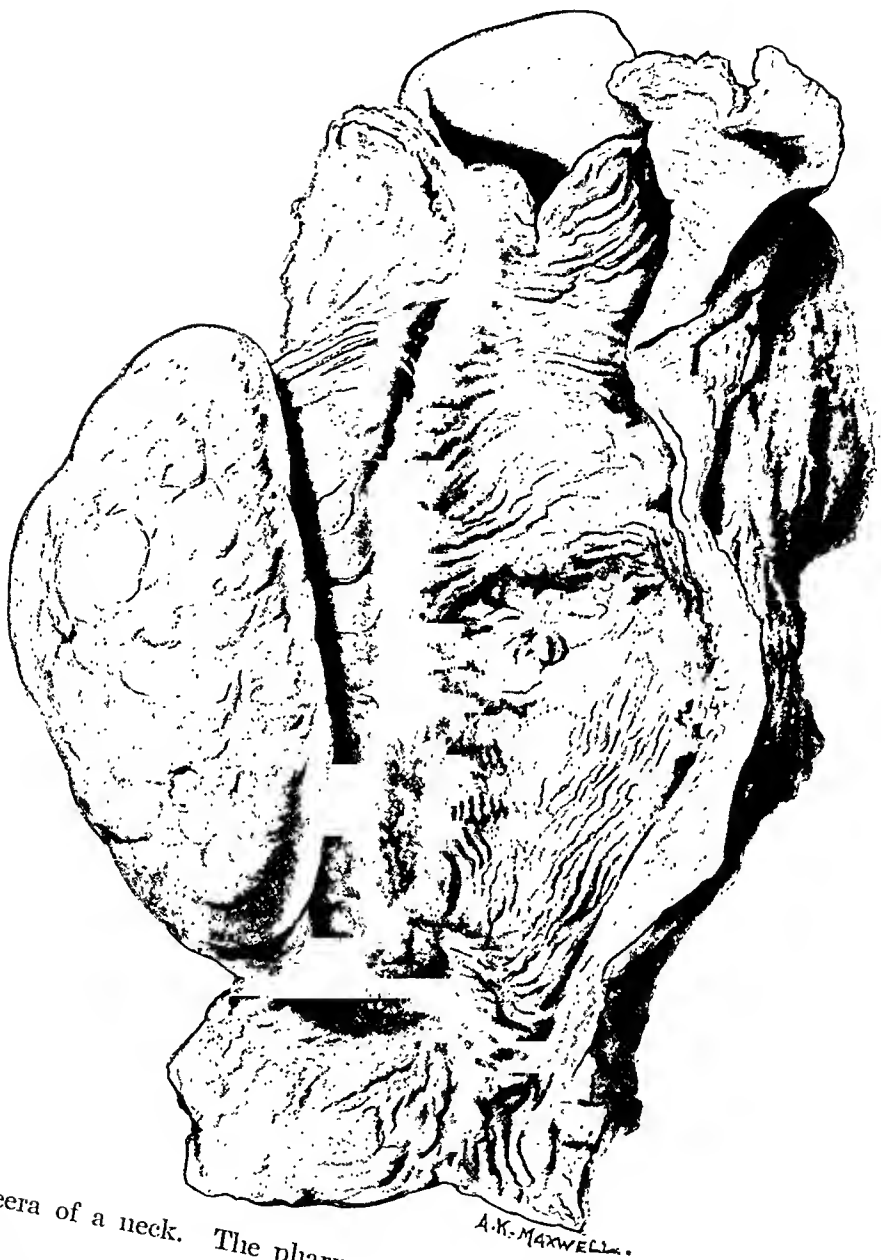
An oval cyst  $1\frac{1}{2}$  in. in its long diameter is embedded in the left wall of the œsophagus and projects forwards by the side of the trachea below the thyroid gland. The recurrent laryngeal nerve was superficial to the cyst.

*Hunterian Museum, R.C.S., 1211.1*

**MICROSCOPIC STRUCTURE.**—The wall of the cyst is composed of a mucous membrane lined by columnar epithelium. Outside this is a double layer of unstriated muscle and then a layer of striated muscle on the surface.

The cyst is probably developed from the 4th branchial cleft depression.

# FIBROMYOMA OF ŒSOPHAGUS.



The viscera of a neck. The pharynx and œsophagus have been opened from behind.

An oval tumour has grown by a narrow pedicle from the posterior wall of the upper part of the œsophagus. The latter is distended, and on its anterior surface is a pressure ulcer through which three tracheal cartilages project. On the front of the specimen the trachea has been opened to show the projection caused by the tumour on its posterior wall.

MICROSCOPIC STRUCTURE.—Fibromyoma.  
*Museum of University College Hospital, 6R1*

No clinical history.



## XLI. CARCINOMA OF THE ŒSOPHAGUS.

(*Malignant Stricture.*)

CARCINOMA of the œsophagus is more common in men than in women and is almost always of the squamous-cell variety with cell nests. Occasionally it is of the basal-celled type. A few cases of columnar-celled carcinoma have been recorded arising from 'islets' of gastric mucosa.

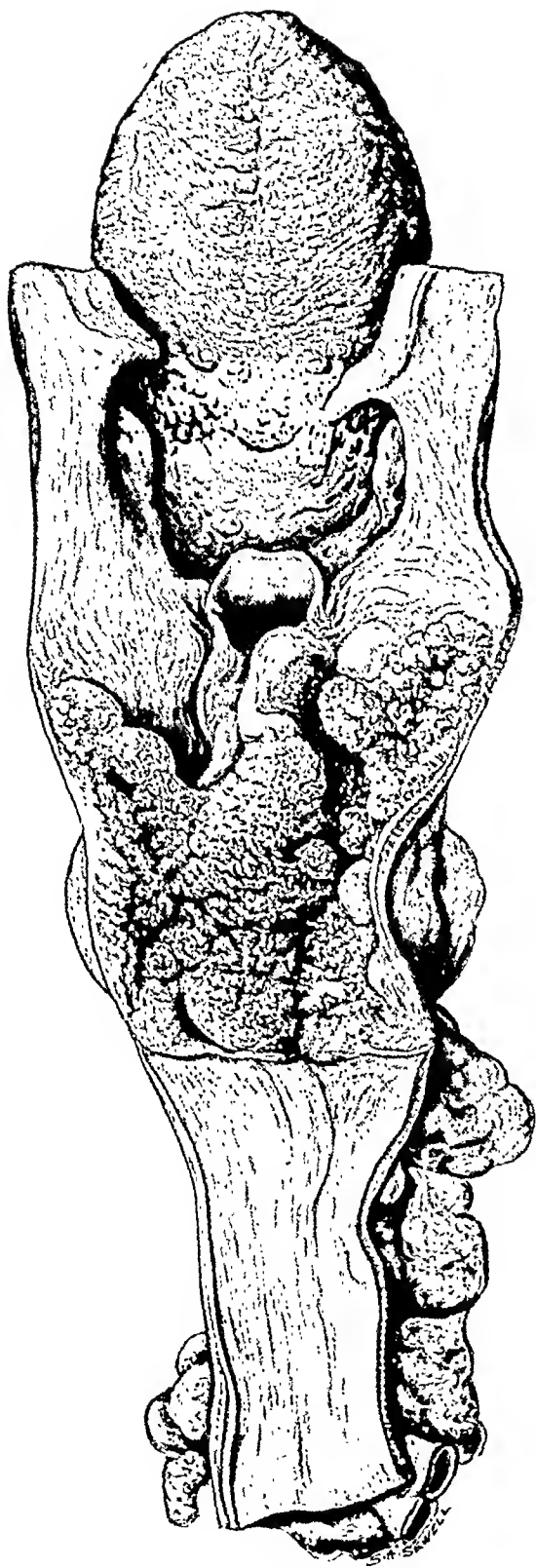
It affects chiefly the narrow portions of the tube and is therefore most often seen at the upper and lower ends and at the point where the œsophagus crosses the left bronchus.

The growth tends to spread round the circumference until it has involved a complete section of the œsophagus. In the longitudinal direction the infiltrated zone may be narrow, but is more often of considerable extent. On the mucous surface the carcinoma forms an ulcer with raised everted edges and an irregular floor. Sometimes a bulky fungating tumour is produced. In all cases there is progressive stenosis at the site of growth. Above, the muscular coat is hypertrophied, but there is little dilatation, since there are no contents to collect as in the intestine above a stricture.

Direct extension of the ulcerating growth may involve the trachea, the vessels and nerves of the mediastinum, or the pleura, and perforation into neighbouring cellular tissue may cause mediastinal abscess. Secondary deposits are common in lymphatic glands, but are rare in distant parts. At the upper and lower ends of the œsophagus carcinoma tends to invade the pharynx and stomach respectively, and, conversely, carcinoma of the cardiac end of the stomach may extend into the œsophagus. At the upper end of the œsophagus the invasion of surrounding structures may give rise to physical signs at an earlier date than the constriction of the œsophagus. Thus the appearance of a hard indefinite lump in the thyroid, combined with hoarseness, may precede difficulty in swallowing.

As in the case of the colon, secondary deposits on the mucous membrane may be associated with a primary growth at a higher level.

Sarcoma of the œsophagus is rare.



## [CARCINOMA OF OESOPHAGUS.

A pharynx and oesophagus with the tongue, larynx, and trachea.

The upper end of the oesophagus and the adjacent part of the pharynx is the seat of a slightly raised and ulcerating new growth. On the right the growth has extended more deeply and has perforated the trachea immediately below the larynx. The tracheal lymph-glands are deeply pigmented, and on the right side are considerably enlarged by secondary deposits of growth.

*Hunterian Museum, R.C.S., 1997.1*

MICROSCOPIC STRUCTURE.—  
Squamous-cell carcinoma.

No clinical history.

## CARCINOMA OF ŒSOPHAGUS.

Four sections of an œsophagus, trachea, thyroid, and surrounding tissues.

In the uppermost section the upper border of a carcinoma of the œsophagus is seen. The growth is infiltrating the trachea and the tissues between the trachea and the carotid artery on one side.

In the next section there is communication between the œsophagus and trachea and a further spread into the surrounding tissues.

In the third section the growth encircles the trachea.

In the lowest section no growth can be seen in the œsophagus, but one lobe of the thyroid is infiltrated, together with a lymph-gland of the deep jugular chain on the same side.

*Museum of University College Hospital, 7R5*

**MICROSCOPIC STRUCTURE.**—Squamous-celled carcinoma.

**CLINICAL HISTORY.**—The patient was a male clerk, aged 56, who suddenly lost his voice while talking to friends eight months before admission to hospital, and had never recovered it since. For six weeks he had brought up mucus from his throat, but had no difficulty in swallowing until shortly before admission. For two months he had had a cough and for one month had been short of breath.

On admission he was emaciated, with a weak and husky voice and frequent short cough. He could swallow fluids only.

The cricoid cartilage and trachea were displaced to the right by a hard mass which moved on swallowing. In the lower part of the left posterior triangle was a hard lymph-gland. The right vocal cord was normal, the left was immobile. X-ray showed obstruction at the upper end of the œsophagus.

He died three hours after a gastrostomy.

**AUTOPSY.**—Purulent bronchitis and myocardial atrophy.

CARCINOMA OF OESOPHAGUS.



MUSEUM OF UNIVERSITY COLLEGE HOSPITAL, 7R5

A.K. MAXWELL.

## CARCINOMA OF ŒSOPHAGUS.

### (PERFORATING TRACHEA.)

The upper third of an œsophagus with the larynx and trachea. The œsophagus has been opened from behind.

For about 3 in. below the cricoid cartilage the wall of the œsophagus is infiltrated with new growth in the whole of its circumference. The lumen is dilated. The anterior wall of the œsophagus at the site of the growth is extensively destroyed, the base of the ulcer being formed by infiltrated peri-œsophageal tissues. At the upper part of the ulcer there is a large perforation into the trachea. The cellular tissue of the mediastinum and right side of the neck is infiltrated by growth and there are nodules in the upper part of the pleura on both sides.

*Hunterian Museum, R.C.S., 6107.1*

### MICROSCOPIC STRUCTURE.—Squamous-celled carcinoma.

CLINICAL HISTORY.—The patient was a man, aged 68, who first complained of hoarseness twelve months before he came to hospital for examination, and the hoarseness was followed by increasing difficulty in swallowing. Latterly there had been occasional pain in the right side of his chest. On examination he could swallow soft solids. There were hard glands on both sides of the neck. The right vocal cord was fixed in adduction and a ragged ulcer was seen in the upper third of the œsophagus by direct inspection.

Treatment by X rays once a week for nine months produced considerable improvement in swallowing and a decrease in the size of the glands.

He died nine months later from broncho-pneumonia following perforation of the trachea.

CARCINOMA OF ŒSOPHAGUS.  
(PERFORATING TRACHEA.)



HUNTERIAN MUSEUM, R.C.S., 6107.1

## CARCINOMA OF ŒSOPHAGUS.

### (PERFORATING TRACHEA.)

An œsophagus with the larynx and trachea. The œsophagus has been opened from behind.

The whole circumference of the œsophagus is infiltrated with carcinoma for  $3\frac{1}{2}$  in. above the bifurcation of the trachea. At the upper end of the growth is a perforation into the trachea measuring 1 in. in vertical length. Below this is a smooth ulcerated surface where the growth has largely been destroyed by radium. The glands on either side of the œsophagus and the peri-œsophageal cellular tissue are infiltrated by growth.

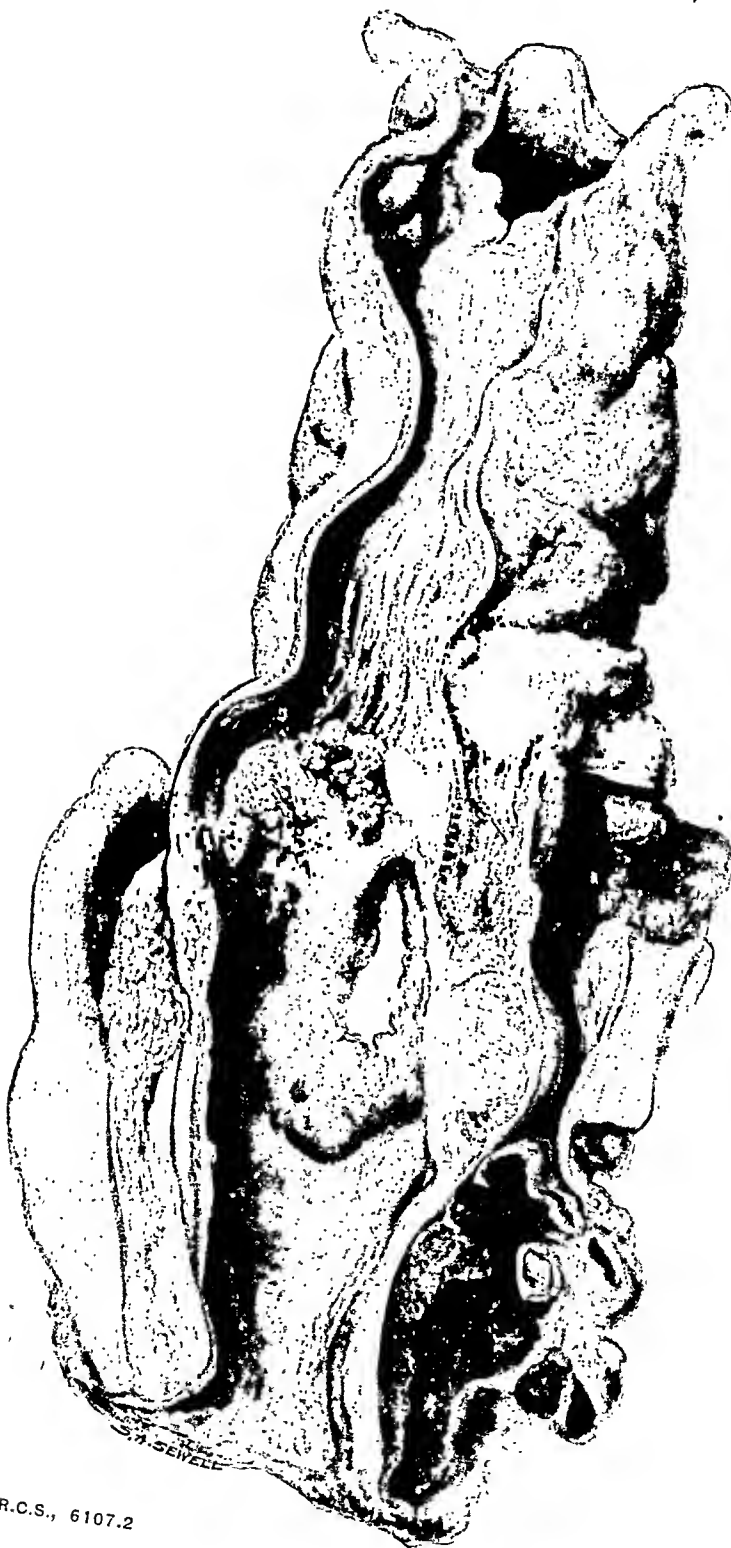
*Hunterian Museum, R.C.S., 6107.2*

MICROSCOPIC STRUCTURE.—Squamous-celled carcinoma.

CLINICAL HISTORY.—The patient was a man, aged 63, who had complained of dysphagia for one month. Œsophagoscopy showed narrowing of the gullet below the level of the 8th dorsal vertebra. Some tissue removed 10 in. from the teeth contained carcinoma. Gastrostomy was performed on account of increasing dysphagia. Four months later a friable bleeding mass was seen in the position of the growth. After treatment by radium the patient was able to swallow light solids for a time.

He died from perforation of the trachea.

CARCINOMA OF OESOPHAGUS.  
(PERFORATING TRACHEA.)



HUNTERIAN MUSEUM, R.C.S., 6107.2



## CARCINOMA OF ŒSOPHAGUS.

Part of an Œsophagus with the lower end of the trachea and the arch of the aorta.

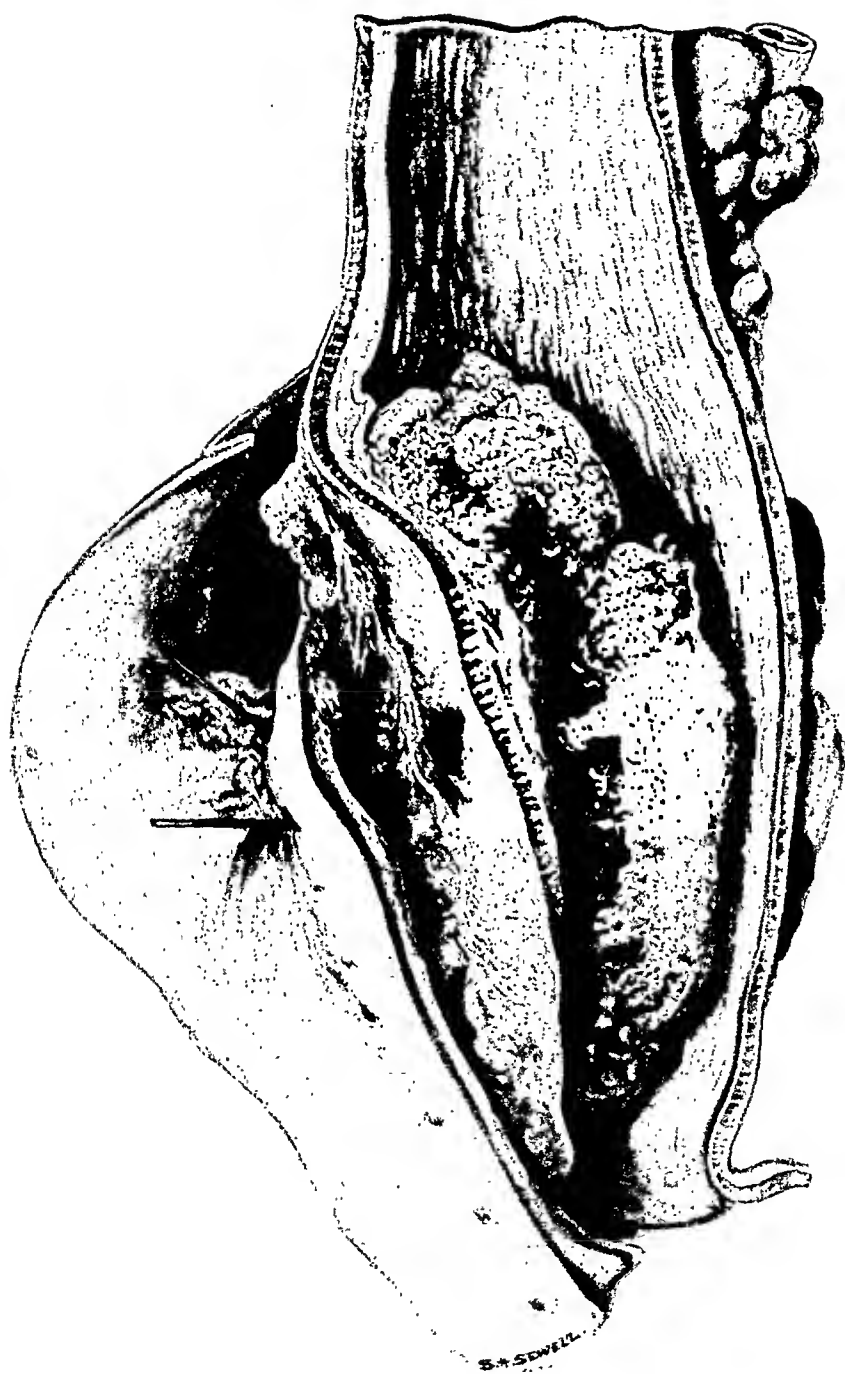
The oval ulcerated growth of the Œsophagus has perforated the aorta. The aperture in the aorta is a slit  $\frac{1}{4}$  in. long between the mouths of two of the intercostal arteries. Its edges are undermined and are turned towards the lumen of the aorta.

*Hunterian Museum, R.C.S., 1998.1.*

MICROSCOPIC STRUCTURE.—Squamous-celled carcinoma.

CLINICAL HISTORY.—The patient was a man who had been attending the out-patient department for some months for carcinoma of the Œsophagus. He was admitted to hospital four hours after a hæmatemesis. Three-quarters of an hour after reaching the ward he had a fatal hæmorrhage from the mouth and nose.

# CARCINOMA OF OESOPHAGUS.



HUNTERIAN MUSEUM, R.C.S., 1998.1

## CARCINOMA OF ŒSOPHAGUS.

(PERFORATING AORTA.)

Part of an œsophagus with the bifurcation of the trachea, the aortic arch, and part of the descending aorta. The œsophagus has been opened from behind.

The incision of the œsophagus passes through an ulcer which involves the posterior and left lateral walls. The ulcer is situated  $1\frac{1}{2}$  in. below the bifurcation of the trachea and is  $1\frac{3}{4}$  in. in length. In some parts the edge is thickened and slightly overhangs the base. In others the edge shelves on to the surrounding mucous membrane. The base is smooth and closely adherent to the descending aorta. Towards the upper part is a circular depression  $\frac{1}{4}$  in. wide with a circular perforation in the middle through which the lumen of the œsophagus communicates with that of the aorta.

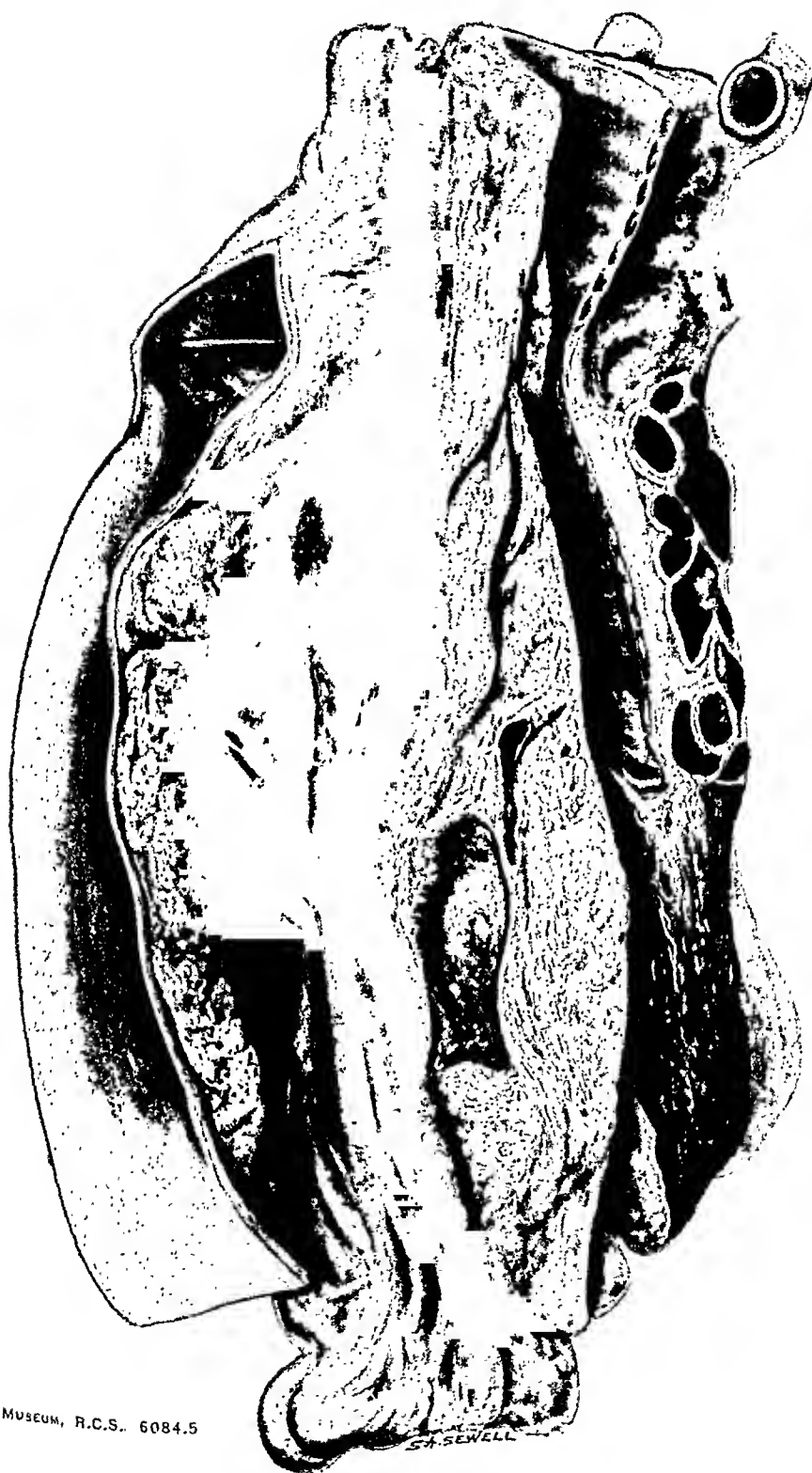
*Hunterian Museum, R.C.S., 6084.5*

**MICROSCOPIC STRUCTURE.**—The base of the ulcer consists of dense fibrous tissue infiltrated with leucocytes, and containing fat in its deeper parts. The surface of the ulcer is necrotic, and beneath are narrow tracts of epithelial cells, polyhedral in shape, extending into its deeper layers.

**CLINICAL HISTORY.**—The patient was a woman who while under treatment for extensive bullous eruption suddenly became pale, and on the following morning sat up, vomited half a pint of blood, and fell back dead.

**AUTOPSY.**—The stomach and intestines contained a large quantity of blood.

CARCINOMA OF ŒSOPHAGUS.  
(PERFORATING AORTA.)



HUNTERIAN MUSEUM, R.C.S. 6084.5

CARCINOMA OF ŒSOPHAGUS.

(PERFORATING BRONCHUS.)



BACK VIEW.

HUNTERIAN MUSEUM, R.C.S., 6106.1

